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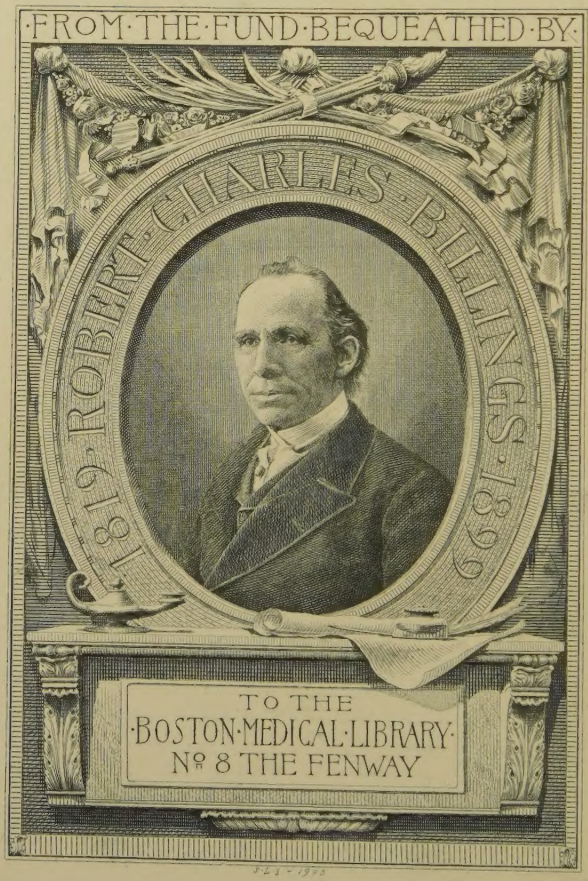

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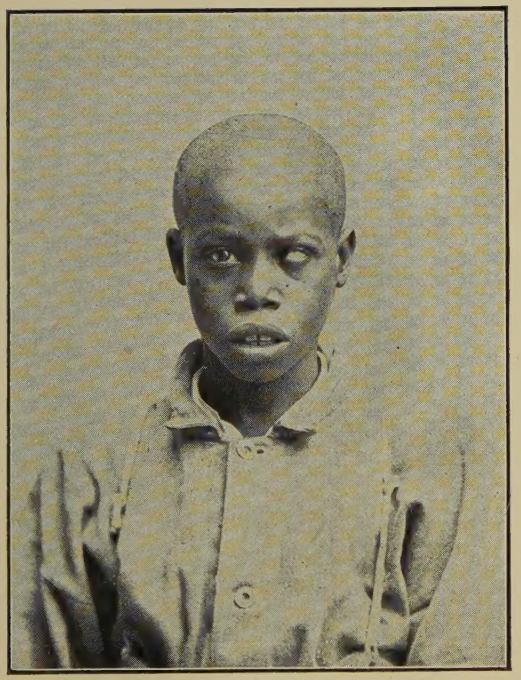

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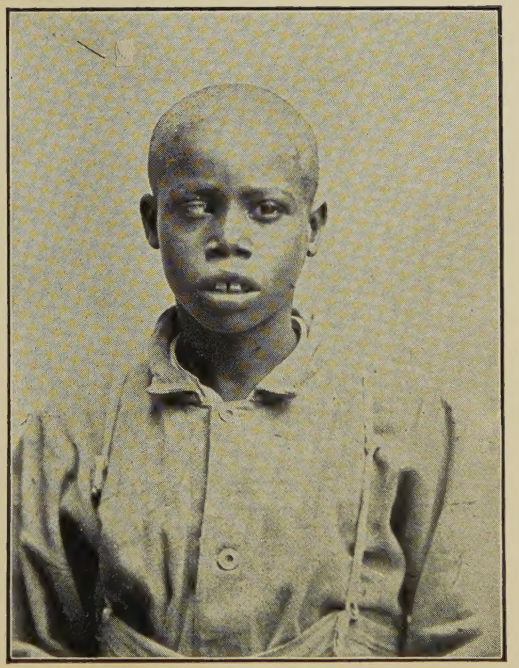

IS IT THE RIGHT EYE?

See illustrative cases, page 132.

These photographs were presented to the author by Dr. Hallock, of Charleston, S. C.

STRABISMUS, OR SQUINT

LATENT AND FIXED

A SUPPLEMENT TO THE

ERRORS OF REFRACTION

BY

FRANCIS VALK, M.D.

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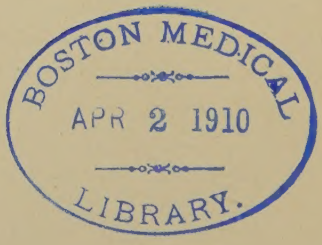

The Knickerbocker Press, New York

To the memory of my Father,

HON. WILLIAM WHITEMAN VALK, M.D.,

SURGEON, 4TH MARYLAND VOLS., U. S. A.,

this work is affectionately dedicated,
in respectful admiration of his work
as a physician and surgeon.

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PREFACE.

I HAVE been repeatedly asked if I had issued a revised edition of my work on the "Errors of Refraction" and have answered that question in the negative for the reason that since that work was first issued my methods of examination of the dioptric media have not changed. But the subject of the imbalance of the ocular muscles—during the past decade—has attracted so much attention from many members of the profession that I have concluded to give the results of my personal experience with cases of muscular imbalance so that my conclusions may be presented to those who are interested in this work. Hence this is partly a supplement to the "Errors of Refraction," and my personal experience in the examination and the correction of the imbalance of the eye muscles; while for a more extended study of the subject I would refer to the many text-books offered by other writers, notably *Maddox*, on the ocular muscles and *Hansell and Reber*, on muscular anomalies, etc.

The motility of the eyes, from that of the normal balance—if there is such a condition—through all the various grades of heterophoria to that of complete and fixed strabismus or squint has been a subject of intense interest to me and while I still think we are hardly past the "threshold of knowledge" in reference to this subject, yet the advancement in this work has been very great, and rapid during the past decade. From my past experience and present work I have formed some views and theories that may not have received the full approval of the profession but they have been very useful to me in a careful study of

my cases and in the final improvement and cure. Perhaps there are many who will not endorse my views, as set forth in these pages, but they are founded on the simple and natural functions of the motility of the eyes and the obvious condition that nature intended in their physiological action, and my opinions have been strengthened and confirmed by clinical work the more I have been enabled to watch and to note the results of this work for the past ten years. I now present these experiences and results—they all relate to one subject, the external muscles of the eye and their imbalance—trusting they may be found useful to the reader. I think I may claim that my method of examination, the indications and the operative procedure have some measure of originality, that will stand a fair and crucial test or criticism and that may be useful in the many cases in which the desired relief has not been obtained by the use of glasses or prisms. I regret that in the preparation of this monograph on squint it has seemed necessary to repeat my thoughts but the conditions are all so intimately related, that in the discussion of any imbalance the same abnormalities and tests must arise thus making the repetition of certain conditions and examinations necessary, I trust they will not weary the reader but serve to impress upon his mind the theory set forth, even if it may not meet with his approval. "That a theory accords with the facts does not necessarily prove it true, although, until some discordant fact is observed it may serve some useful purpose." I believe that the action of the ocular muscles, in their relation one to the other, in reference to the fusion power of the eyes and the physiological processes that govern their movements is not fully understood, and trust this work will, in some way, assist others and stimulate them to further and more useful research in this most interesting field of ophthalmology. I have quoted

the writings of many others in this work and am thankful for the benefit these writings have been to me and for the liberty of quotation. I wish to take this occasion to gratefully acknowledge my early training, in the action of the ocular muscles and their standard test with prisms, by my friend and first instructor Prof. D. B. St. John Roosa, in whose work I have always felt the deepest interest, for the past twenty-five years. To my associates and assistants Dr. E. M. Alger—for his revision and approval—and Dr. Gertrude Allen, I owe many thanks for their suggestions and assistance in the preparation of this work.

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Strabismus.

CHAPTER I.

ARGUMENT.

If there is any reason why this treatise should be offered to the profession of medicine at this present time, it is in the fact that there seems to be no settled theory as to why the eyes should deviate or tend to deviate from the natural position of the visual lines as evidently ordained by nature. That they do deviate or have a tendency to that condition is a self-evident fact, clearly shown in numerous cases; but why does an eye that should be normal assume or tend to assume an anomalous position? This question has never been satisfactorily nor successfully answered. Some eminent men have advanced their theories of squint, either latent or fixed, but they all have met with opposition and have not proved reliable from the fact, that while having many arguments favorable to their theories, yet we see many cases, in active practice, that seem to disprove these theories completely. Furthermore, why is it that among all these theories, advanced in reference to the etiology of strabismus or squint, no two of them seem to come any where near an agreement? One theory is advanced that will have many followers; another theory seems to find the same number of admirers, and in simple justice I must say that my own theories on this subject have met with many objections. Yet as I read the text-books of to-day I see many suggestions that point to the muscular or anatomical theory of squint.

There must be one underlying cause, even though we may have so many contributing causes, and, just at this point, may we not attribute the reason for such a vast difference of opinion to the fact that one may very readily consider a secondary, or contributing cause as the primal one, notably so in Donders' Antithesis, yet careful examination, by the most advanced methods of the present day, will demonstrate one condition always present in every case of deviation of the visual lines. If so, it seems reasonable to me, that such condition must be the one true and prime cause of all cases of Strabismus, either fixed or latent and in any and all directions.

Theoretical considerations of this subject will not do. The eyeball is not a camera. Its action as to rays of light differs from that of a box or lens. The ocular muscles are not the "reins to drive a horse" or simply to mechanically move the eyeballs in and out and up and down, but these beautiful anatomical structures of the eyes are controlled and adjusted according to the laws of nature and also controlled by the human being "behind it all" that must influence them in many ways. Then behind all this beautiful, complex, and intricate system of tissues, (nerves, muscles and vessels), we find the most simple rules and observations to point out the movements of the eyes and the action of the muscular apparatus. Let me illustrate this in the action of the obliques. According to our text-books these muscles seem to play a very important part in the economy of the movements of the eyes in the field of rotation and yet, we may ask ourselves if the same movements might not occur if the oblique muscles were absent? If so, then why shall we consider them at all? Well we need not in a practical sense, but at the same time we must remember that the adjustment of the eyes in the orbits and that of the vertical plane of one eye to that of the other eye, is so exact

that any anomaly of one or more of the straight ocular muscles, either due to an anatomical condition or to a paresis, partial or complete, must disturb the harmonious action of all the others, in a greater or less degree. So, in the case of any deviation or tendency to deviate we must keep in mind this beautiful adjustment of our visual lines and the vertical planes so that we may always produce that priceless function, binocular vision.

Then in the study of all these functions of the human eye, these "windows of the human soul" we must consider them as acting, not as a human machine, but as one of the most beautiful and complex systems of nature, and yet, one that may be demonstrated as simple and easily understood if we will only look at the subject in the light of nature and of her work. As we study the physiological properties of the eyes and their muscular apparatus, is it necessary to consider them as moved downward by the combined action of two or three muscles or simply by that of one, the inferior? We may suppose that all of these muscles act in the function of looking downward, and perhaps they do, for I doubt very much that nature ever placed a useless organ or muscle in the body, and the action of these muscles that may be accessory to the action of one muscle may be necessary, but to my mind are not. Furthermore, to continue the argument: A paralysis of the superior oblique (which, by the way is very rare) will seem to interfere with the function of looking downward, as well as in all other directions, but is not this fault due more to the disturbance of all the actions of the ocular muscles from the paralysis of such an important muscle as that of the superior oblique? A paralysis of any ocular muscle must disturb the action of all the others in a greater or less degree but I think that in all cases under a carefully-conducted examination we can locate just what muscle is or may be affected.

The subject of heterophoria or of squint is attracting the attention of many students, and with our increased facilities for the examination of the refraction and the musculature of the eyeball that we have at present, we should know and understand what is the prime cause, what lies beneath all the more superficial conditions, and when we do locate this cause I think we may find a reason for *all* cases of strabismus or functional deviation of the visual lines. Not a cause that will cover some of the cases but one that will cover all and every case. We may then paraphrase Lincoln's celebrated saying: "Some of the cases may have one cause, one cause may cover some of the cases, but we must find one cause that will cover all of the cases," and we shall then come closer and closer to nature's everlasting truth.

CHAPTER II.

THE MOVEMENTS OF THE EYE.

It is an easily demonstrated fact that the eye must remain perfectly still in any one position when the function of vision takes place, and as a corollary to that we might say that the eye is practically blind when moving from one position to another, inasmuch as no images can be formed on the retina as the eye moves from one point of regard to another. Dodge says "the field of clearest vision is practically a point, namely that point at which we are looking;" but I should be inclined to designate this point as a region whose extreme boundaries, though not perfectly defined, would form an angle with the nodal point of the eye of about four degrees, and all objects lying within this region will be clearly and distinctly seen. At the same time, this region seems to have a central point which tends to regulate and control the fusion of all images and the fixation of the eye. This is the point at which the visual line ends, as it is extended through the nodal point from the object looked at. Now this region of most distinct vision is evidently that of the *macula lutea*, or yellow spot, but is more or less extended on the retinal field, and in space, at a distance of one foot from the nodal point of the eye, will include a region of about one inch in diameter. Within this region vision is perfectly normal at the reading distance.

It is very evident then that the eye could have no continuous motion from one side of the page to the other, that is, from left to right, but must pass by little jerks, as it were,

and periods of rest, from one point of regard to another, so that as we read a line across the page the eye is practically at rest much longer than it is in motion. Similarly, we have the very interesting experiment of Stratton, as to the movements of the eye in following a curved line, like a circle. Here the movements of the eyes were very irregular, but always passing from one point of regard to another by rapid movements and minute periods of rest, as described above, he says, "this path by which the eye passes from one to another of these resting places does not seem to depend very nicely upon the exact form of the line observed," it is extremely irregular in outline, whether the object, as a curved line, be artistically made or very irregular. The same interesting movements take place even when the eye is moved from point to point in space, but the movements of rest are so slight that the retinal impressions do not reach the higher centres of the brain, unless the impressions be very sharp and vivid. To illustrate this, if we stand in a darkened room and look at the electric spark of the static machine at a distance of six feet, and then slowly move the eyes from right to left, we will see a series of minute bright spots, with dark spaces appearing between them about two or three mm. apart.

It must be evident from this that the eye during its excursion from right to left must have made a series of minute stops sufficiently long to allow this intense image to be formed on the retina and so conveyed to the brain, also that no image was formed while the eye was in motion, as shown by the dark spaces between the brilliant spots. These phenomena have been long observed by some of our investigators in the field of physiological sciences, and during the past two years have been more fully investigated and proved by means of a reflection from the cornea and a photographic plate.

Raymond C. Dodge, Stratton and others, have made many investigations in these interesting phenomena, by photographing a bright reflection on the cornea, as the eyes are moved in reading, or as the look passes from any point of regard to another. If we attempt to pass the look slowly from one point to another, so as to see clearly all the objects that may be present between these two points, and another person closely watches the eye movements, or a photograph is taken of the reflection from a bright spot on the cornea, it will be seen that the eye has made several rapid movements and stops from point to point across the field of motion. This cannot be done introspectively as we are not conscious of these movements of the eyes any more than we are of the similar movements of fusion in the act of binocular vision.

My conception of this physiological act is that the movement of the eyes from one point of regard to another is purely a voluntary one, controlled by the higher brain centres, while their action in the fusion of the images according to physiological law is controlled by the lower or automatic brain centres.

Furthermore, under this voluntary movement of the eyes, as the look passes from one point of regard to another, sufficiently far apart, we are practically blind during the period of transition. In other words, we are not conscious of any object in the line of movement, though we may know that the most sensitive part of the retinal field must have passed the rays of light from such objects. It is true that we may be conscious of these objects, but unless we were familiar with their appearance we could not tell what they were; consequently when the eyes pass from two distant points, say ten feet from the eyes, and separated fifteen feet, we see the two points clearly, but not the intervening objects; and if we attempt to move the eyes slowly between these two points, so as

to see all the objects between them, the eyes will make a number of stops and rapid movements of which we are totally unconscious.

Dodge has proposed five type of eye-movements in the horizontal meridian, as those of Fixation, Pursuit, Co-ordinate compensation, Reactive compensatory, and lastly, Unique movements, which seem to me are the movements of fusion. The first three are very important in connection with this study and show how extremely delicate and perfect is the adjustment of the visual line by the action of the muscles of the eye. The first type, Fixation, are reactions to eccentric retinal stimulation that seem to be dependent on a physiological function from birth and are not influenced by voluntary effort. The second, Pursuit movements, are the involuntary movements of the eye as we look at an object from a moving train, in which the "line of regard seems to lag behind the line of interest and to overtake it from time to time by short eye movements of the first type." The third type is of interest as showing the constant fixation of the visual line while the head is moved slowly from side to side.

Rudolph Panse, Archives of Otology, on Vertigo: says, "we judge of the position of objects laterally by the degree of ocular movements, which in turn is determined by the amount of innervation supplied to the muscles. This estimation takes place through nervous processes of which we are unconscious. If the ocular muscles should be weakened the necessarily increased innervation, for its movements gives the impression of a greater excursion."

I have dwelt thus briefly on the various movements of the eyeballs in the orbits to show how delicate and yet how exact all the physiological movements of the eye in the adjustment of the visual line must then be in the function of perfect binocular vision. Then the question of greatest importance is the adjustment of the visual line

and the anomalies that may be present in the muscles by which these functions are produced, and by what conditions they may be disturbed. By these questions we are brought to a consideration of the ocular muscles, their action, their size, and their insertion into the sclera.

In the ocular movements, both voluntary and involuntary, we are not concerned—when studying strabismus—as to just how many muscles may participate in any one certain movement. These functions are referred to in our text-books, but in this study we shall only consider the action of the interni in the inward movement, or the externi in the outward movement, and leave out whatever participation the superior and inferior may have in the lateral movements of the eye. We shall also omit the action of the oblique in the upward and downward rotation. These oblique muscles only tend to keep the vertical meridians parallel with each other, and have no connection with our consideration of the subject of squint. In making this statement I am well aware that the investigations of Stevens and Savage would lead us to the very complex study of an insufficiency of the oblique, but the participation of these muscles in the anomalies of the straight muscles has not yet been fully demonstrated, nor have I met with any cases where a weakness of the obliques could possibly be shown unless there was some pathological condition present.

I do not propose to demonstrate the anatomy nor the physiology of these muscles, except so far as we may wish to understand their action in our daily work nor shall I try to form any special theory, or follow the text-books on this subject, but simply to illustrate what nature in her beautiful work intended as the functions of the eyes and the muscular apparatus. Perhaps I offer a theory, but if so it is one that has been suggested to me as I have examined my cases in daily practice. Clinical work in

the hospital gives us some very valuable suggestions, but few have the time—in the rush of a clinic—to fully study the various phenomena that may be noted. But in our office work, where the individual responsibility comes upon us, we must stop to think of the points that develop as the work proceeds, and to ask ourselves why certain phenomena occur.

In trying to find an answer to these questions that arise, I have endeavored to understand what was the natural action of these muscles, or in other words, what purpose did nature—in the full understanding of all her requirements—intend that the eye muscles should perform in the natural course of life. Let me start out with the statement that in this study we will not consider the origin of the ocular muscles, they all come from practically the same region, except the inferior oblique. Leaving out, then, from our consideration, the complex movements of the oblique muscles, it is with the straight muscles of the eyes, their insertion into the sclera and their size and weight, where we shall find the most interesting points and by which we may understand what nature intended should be the normal action of the ocular muscles and the reasons for the various anomalies that may exist. In the anatomy of the other parts of the body, as the muscles of the extremities, we find that the needs of the body have been fully provided for, as the parts that require the most power and action have the largest and most powerful muscles. Furthermore, if we compare the anterior with the posterior aspects of the arm or leg, do we not find one much more developed than the other? then we may justly look for the same wise provision of nature in the eyes. They are the only parts of the human body that never seem to be at rest, as it is claimed that the eyes move even during profound sleep.

Anatomically, our first consideration will be the insertion

of these muscles into the eyeballs and Fuchs' results may be accepted—even though we may find great irregularity in many cases—as I believe his results will be found correct and to my view they seem to point out nature's intentions. Quoting from Norris and Oliver's System, we have, first, the distance of the insertions of the ocular muscles from the cornea and Fuchs' results give us these figures :

Internal rectus, = 5.5 mm.
Inferior rectus, = 6.5 mm.
External rectus, = 6.9 mm.
Superior rectus, = 7.7 mm.

If the assertion is true that the nearer to the cornea we find the insertion of a muscle, the greater do we find the power of that muscle to turn the eyeball, then a glance at this table will show the relative power that nature intended should be exercised by those muscles. But let us continue our investigations anatomically a little further and see what is the relative gross weight of these muscles ? and from the same authority, we note Volkman quoted as follows :

Internal rectus, = .747 grammes.
Inferior rectus, .671 "
External rectus, = .715 "
Superior rectus, = .514 "

Again a study of these figures will give us the same results as we had in the insertion of the muscles, as we find the greatest weight, as well as the most advantageous insertion, is according to the daily needs of these muscles for action. Hence, we have here two very important factors that seem to show the natural action of the straight muscles of the eyes.

What are the movements of the eyes—in the function of binocular vision—that seem most necessary in our

daily life? First, the lateral movements, associated with convergence, and so we may expect and find, that the muscles that produce these movements will have the greatest size and the best insertion, furthermore, as convergence is an addition to the lateral movements, we find that the internus is the largest in size with its insertion nearest to the cornea. Next to that muscle is the externus, that can only turn the eye outwards. Then we have the inferior, as we are required to look downward much more often than upward, and lastly we have the superior, that we find is the weakest of the straight muscles of the eye. Now if this is nature's anatomical arrangement of these muscles, do they not convey a certain relation to each other in the necessities of life? and if so, may we not ask what is this relation?

Thomas Dwight, M.D., LL.D., in Norris and Oliver's work, says: "Practically, it is to be remembered that it is very unlikely that one or even two of these muscles ever act alone. The accuracy of any movement is due, not alone to the pull of the muscle to which it is usually ascribed, but also the graduated resistance of the antagonists. Probably the simplest movement of the eye is made by the more or less active concurrence of all the muscles." This seems very well in theory, but can it be proved by any actual demonstration on the living subject? And if it is only theory, will not the supposition that each muscle has its particular function in the human economy of the eye do just as well, and at the same time assist us very much in the study of our cases and the means of relief, if needed? I am inclined to think so, and will endeavor to give my conception of their action.

The movements of the eyeballs about their centres of rotation is a subject that has been so freely discussed that a further consideration may not be necessary, but, at this time the action of the ocular muscles and their deficiency

in power is attracting a great deal of attention from the profession, and so the individual opinion of one who has studied the subject somewhat may be of service. I am inclined to simplify the action of these muscles so as to give each one its action in a very simple manner and to discuss how they will act, either singly or combined, by their associated movements, their voluntary and involuntary action and to entirely separate the action of the obliques from that of the straight muscles.

If one will read of the action of these muscles in Fuchs' Text Book of Ophthalmology, page 576, he will find one of the best descriptions, and I would refer the reader to that work, but I think that each muscle receives its innervation from the nerve centres, as it is necessary to turn the eyes, and a careful study in this direction will show that there is no necessity, nor proof that the eye will require the action of three muscles to turn inward, etc.

All these muscles act by their natural tonicity to keep the eyes steady in a fixed position when at rest or in the first position. As simple antagonists when the eyes are moved from that position, and by their innervation the eyes can be turned to any point by the straight muscles alone. Can we not turn the eyes inward, as far as necessary, by the action of the interni; outward, by the externi; upward, by the superior and downward by the action of the inferior? while the eyes can be turned to any point between these positions by the combined action of these muscles, as upward and inward by the interni and the superior recti and so on in all other directions without the muscular action of the obliques? Perhaps our text-books do not say so, but the above proposition is reasonably true and assists us very much in the study of the correction of an imbalance of these muscles. Accepting this proposition, then we can leave the movements of the eyes to the

straight muscles alone and find that the oblique muscles will receive their innervation to turn the eyeballs on the sagittal axis so as to keep the vertical meridians of each eye parallel to each other. This function of the obliques is entirely involuntary ; we cannot cause them to act alone by any power of the will, but they are controlled and stimulated by the centre for fusion, or the blending of the images on the retina, so that the image of all lines in the vertical meridians shall fall upon corresponding parts. In other words, the action of the obliques simply tends to keep the vertical meridians of the eyes parallel to each other and does not assist in the movements of the eyes in the field of fixation.

Now the eye is one of the most perfect instruments in the human economy, in which the beautiful adjustment of focal power will bring all rays from an object so as to form an image on the retina, with each image falling upon corresponding parts by the action of the straight muscles of the eye, controlled by the fusion force. These rays from the object pass through the various media of the eye and are refracted so as to form the image, and in these rays we find one central ray, passing from the center of the object to the center of the image on the retina without any deviation whatever, and this ray forms practically the visual line, all other rays being refracted, except those forming the secondary axes.

If nature has provided for this beautiful adjustment of the vision, then I think we can trust her in the movements of the eyes for the purpose of binocular vision and bear this in mind when we attempt to study the different actions of the eye and the muscles by which the eyes are moved in various directions. May we not ask the questions, first, what is nature's object in the movements of the eyes? and secondly, how has she provided for those movements that are necessary for our well-being? Shall we study

them simply from her standpoint, or shall we put the eyes in a position that nature never intended they should be in, and then try to find out what is the action of the straight muscles or that of the obliques? I am led to this inquiry as I find many observers seem to draw their conclusions from certain tests after they have put the eyes in a condition that is practically abnormal.

To be more explicit we are told that we may find a paresis of the obliques by turning the eyes to their extreme limits, in certain directions, and there find a tipping of the image, while on the other hand, when the eyes are in their normal position we will not have this tipping in any direction. It is the same in the various tests that have been suggested to us for a deficiency in the power of the straight muscles, as by the vertical diplopia test, we entirely destroy the fusion power and then ask, if the muscular power is below normal standards. The same objection applies to the tests with the Phorometer and the Rod.

If these objections are practical, shall we not keep the eyes as nature intended them to be and then endeavor to find out if there is any fault in the muscular action? It seems so to me, and from that standpoint I have tried to base all my conclusions as regards the action of the ocular muscles. We have then two natural functions in the movements of the eyes; one that is principally under the control of the will, the power to direct the eyes to certain points, a fixing power, and one that is not under the control of the will, automatic, the fusion power. The first function tends to keep the visual lines fixed on the object and the second tends to keep the images on corresponding parts of the retina. These functions are controlled by different parts of the brain and to them we may add, the function of convergence controlled by a special center.

Hence, in the muscular system of the eyes, it seems to me we have three forces acting, that are partly voluntary and partly involuntary. In the first action we have the power to move the eyes in all directions as the will may direct, the field of fixation, a power that is controlled entirely by the action of the will-power of the brain. This power enables us to look in all directions by the action of the four recti, acting either singly or in combination with one or more of the other muscles, and the action of these muscles is controlled by the centers of motility of the eyes. Secondly, we seem to have the power to move the eyes—one independent of the other—by which we are enabled to keep the images fixed on the macula of each eye. This power seems to be an involuntary one, as we cannot control it by any power or action of the will. This is the fusion power of the eyes or as Savage so well puts it, *the guiding sensation*, which seems to be controlled by a different center of the brain than that which controls the motility of the eyes. Lastly, we have the power of convergence, also controlled by a separate centre of the brain—well shown in paralysis of convergence—which acting on the interni from the first position to that of extreme convergence gives binocular vision of all objects situated within infinity.

In the study of the action of the ocular muscles, the *fusion* power of the eyes is of the first importance. I notice that it is practically ignored by most writers on the motility of the eye, yet in all our clinical work it is a most important factor, as it is so absolutely necessary for the function of binocular vision, as we must have perfect fusion of the images, on their respective retinas, to have perfect binocular vision both at the near and distant points. We may separate these images mechanically—as I shall show by the action of prisms—yet if we do separate them, they will again fuse by an involuntary act controlled by some

center of the brain and not simply by the power of the will, until they are so displaced that the natural power of the muscle is not great enough to cause the images to fuse, in other words, the muscular system may not be strong enough, under the stimulation of the guiding sensation, to move the eyes so that both images will fall upon the macula or on corresponding parts of each retina.

As nature "abhors a vacuum," so does she naturally object to the double images or diplopia, and when we do produce diplopia by displacing the rays of light from an object, by passing them through a prism, the brain becomes aware of the diplopia and seeks to obviate it by turning the eye on its center of rotation until we again have single vision. This can only be controlled by the innervation of the ocular muscles and this innervation can only act on the muscles according to the relative power to turn the eye in a certain direction. If I deviate the rays from a candle outward, then the image is formed on the outer part of the retina and diplopia results, but at once, as the fusion power is disturbed we find the eye turning inward, until single vision is again produced. Provided, the power of the internus is great enough to turn the eye inward until the deviation of the rays is overcome and the visual line will correspond with the central rays as they pass through the prism. But, if the rays are deviated so far outward that the internus cannot turn the eye sufficiently inward then no power of the will can fuse the images, and diplopia remains.

This fusion power of the eye is then an involuntary action, not only on the straight muscles to keep the image fixed on the macula, but also on the oblique muscles, by which we are enabled to keep the vertical meridian of each eye parallel so that the other parts of the image, will fall upon corresponding parts of each retina, as long as

the visual line remains within the limits of the field of fixation.

Before we proceed further, with the tests of these ocular muscles in reference to their power to move the eyes, it will be understood that all degrees, as represented by prisms, refer to the angle of the prism and not to its deviating power, unless so expressed. As a simple rule, we may understand that the refracting angle, or power to deviate the rays from an object is one-half that of the angle of the prism.

Proceeding to a further study of the action of these muscles, we may note: first, for the purpose of illustration, the field of vision, which, as measured by the perimeter, with a small, square white object, is outward, 90°; upward, 50°; inward, 60°; and downward, 70°; these measurements may vary somewhat in different individuals. This being the extent of the physiological limits, in which the eye can distinguish moving objects, when in the first position.

In the same manner we find the field of fixation, which is represented by the extreme excursions that the eye can make in all directions while the distinct image of the object will be fixed on the macula. We know that the macula region of distinct vision—and by distinct vision I mean the ability to see two very small dots on a white card and close together, or the ability to read No. 1 Jaeger,—is very small; and its region in space about one inch in diameter on the page of a book, held at the usual reading distance. Then to find the field of fixation, we test the eyes in reference to the ability to see these two small dots, or to read the finest type, clearly, in all directions. We find that this field is represented by the ability of the eye to turn outward. I make this statement as it seems to me that the field of fixation inward is limited for each eye, in this test, by the contour of the nose;

we see clearly in the inner part of the field until the object passes behind the prominence of the nose.

The field of fixation may then be expressed by the power of the straight muscles of the eye to turn the visual line in all directions, with clear and distinct vision of the smallest objects.

DeSchweinitz gives the limitations of this field or the excursions of the eyeballs, at about 90°, vertically and laterally: with downward rotation, 60°; upward, 30°; inward, 45°; and outward, 40°; while an average of Duane's measurements gives the rotation, as downward, 60°; upward, 43°; inward, 47°; and outward, 46°; these results are about the same as found in my own measurements of the same field, using the finest type and at the farthest distance from the centre of the perimeter, that it could be read distinctly.

Stevens' measurements with the tropometer, seem to be the most practical and exact, as this is essentially an objective examination depending on the excursions of the eyeballs. Measured by this instrument the field of fixation is very nearly the same, as we find the excursions of the eye, downward, 50°; upward, 33°; inward, 55°; and outward, 50°; all these measurements being practically the same. There is very little difference between the objective and the subjective fields of fixation. This power to turn the eyes in any direction—limited by the power of the straight muscles of the eyes—is purely voluntary, and under the complete control of the will as far as binocular vision is concerned, as in these tests, the co-ordinating power of the eyes is the same whether we cover one eye or not, and the monocular field is usually the same in each eye, *unless* we have some anomaly in the action of these muscles present, as an insufficiency of power or a paresis. These movements of the eyes have been lately termed *version*, in contradistinction to that of *duction*,

and may be designated as inward or outward version, and so on. I will now call your attention to what is one of the most important functions, a third field of vision in the examination of the eyes, which we will term the *field of fusion.* This field is represented by the excursions of the eye to fuse displaced images on the retina in any *part* of the field of fixation. This is a physiological function that seems to be almost involuntary, and that must be controlled by a different centre of the brain from that of the excursions of the eye in the field of fixation, or at least, the muscular sense seems to be acted upon by a special centre different from that which innervates the movements of the eyeballs on the centre of rotation in version. The designation of *duction* to this function is very appropriate, and it can be referred to as adduction, abduction, sursumduction and deorsumduction.

In reading Morgan on Comparative Psychology, in the chapter on Automatism and Control, I am inclined to think that this fusion power of the eyes is controlled by the lower centres of the brain and is automatic in its action, and not by the higher centres under the control of the will. . . Be that as it may, the centre for fusion has not yet been demonstrated, but it seems to me there must be some control over the movements of the eye to produce perfect binocular fusion of the images. All of which remains to be proved. DeSchweinitz, page 507, says, "fusion is believed to be the origin of the impulse which directs the movements of the eyeballs, especially in association in the same direction." If this be true—and I firmly believe it—then we must change many theories of the movements of the eyes and our tests for insufficiency of the ocular muscles, also, in my opinion, some of the theories of the causation of strabismus. I referred to this fusion power in reference to squint, in a paper read before the New York County Medical Society on "Crossed Eyes;

How shall we treat them ?" November 24th, 1894. Fuchs, in his text-book, speaks of this function as the "tendency to fusion" due to "attempting to bring the eyes by suitable muscular effort into a proper position, so that the double images coalesce" and illustrates it by the power to fuse the images when they are separated by the deviating action of a prism. Hence as I understand it, the field of fusion has no reference to the movements of the eyes within the field of fixation, in fact, seems to be completely independent of the normal ocular movements of the eyeballs and is practically dependent on the relative power of the straight muscles. Then the power to turn the eyeballs on the centre of rotation has nothing to do with this fusion power, as each function seems to me to be separate and distinct.

To repeat, what are these two functions of the eye, and are they not controlled by different centres of the brain ? First, we have the power to turn the eye, on the centre of rotation, in any direction by the innervation of the straight muscles of the eye, which in all persons is a fixed power, as shown by the field of fixation, as all the examinations are about the same, as inward and outward, etc., the limit generally between 40° and 60°. On the other hand, the excursions of the eyes to produce fusion of the images is very much smaller, the muscles must receive their impulse from a different centre of the brain and the limit of turning the eyes, so as to fuse the images—fusion power—is evidently controlled by the *relative power* of the straight muscles, which is the same in all positions of the eyes in the field of fixation.

If we turn the eyes to the right 20° ; or to the left 20° ; or upward or downward, we still find this fusion power the same in all positions ; adduction still has the same relative power in reference to abduction in one part of the field as the others, as the images will fuse with the same

prisms placed over the eye as they will in the primary position.

What is this relative power of the straight muscles of the eyes? Is it not represented by the normal, physiological action, anatomical construction and insertion of these muscles as shown in the first part of this paper? Are not the natural needs of the eyes exactly represented by these muscles as they act in the field of fusion? I believe so, as we are required to seldom turn the eyes upward; more often downward; and constantly in a lateral direction, with which we must associate convergence; and the limit of the field of fixation upward, demonstrates that the weakest muscle of the four recti is strong enough to turn the eye on the centre of rotation to the extreme limit of fixation, while the fusion power to turn the eye upward is only 3° more or less.

Hence, the relative power of these muscles has no relation to the power to turn the eye in the field of fixation, and consequently has no reference to the power of version, as usually suggested, but simply to the power of fusion. In other words, I can turn my eyes to the limit of fixation, or say 30° to the right and still fuse the images separated by a prism, according to the relative power of the straight muscles, the same as I can when the eyes are in the primary position.

From the preceding remarks, we must have certain parts of the brain that seem to control the movements of the eyeballs; one that controls and stimulates the movements of the eye in its excursions, that is, that controls the action of the four recti. which move the visual line within the field of fixation, the centre that controls convergence and the centre that controls the action of the oblique muscles to keep the vertical meridians of the eyes parallel, simply turning the eyeballs on the sagittal axes. These centres control the movements of the eyes

to any point in the field of fixation and independently of these centres we have the function of the brain that is stimulated by the guiding sensation of the eyes to preserve binocular vision and to prevent diplopia—within the field of fixation—and that innervates the eye muscles according to the extent of the field of fusion.

What is the extent of this field of fusion, and how shall we estimate it, as it varies in different individuals? This will depend upon the power of the muscles—one independent of the other, according to the stimulation of the guiding sensation—that can be represented by a prism, which will deviate the rays of light from an object so as to fall on some part of the retina, distant from the macula, and yet can be fused or blended by muscular action. This momentary diplopia will stimulate the centre for fusion and so innervate the ocular muscles to produce single vision. The strongest prism with which we can produce fusion represents the power of the eye to move within this field. I think it best to make all these tests from a distant point, as twenty feet, so that the eyes may be free from the stimulation of convergence with accommodation and the increased illumination of the near point. Hence I place the candle or other illuminated object at a distance of twenty feet from the patient and a little lower than the plane of the visual line when fixed, so that the eyes may be in the primary position. Then place a good frame on the face and in this place a prism with the apex over the muscle to be tested. In this manner we note the extent of the field of fusion in all directions.

In proceeding with this test we should give the guiding sensation time to act on the innervation of the ocular muscles, commencing with the weaker prisms and increasing their strength as long as the images will fuse or blend into a single image of the object. These tests will vary from day to day and no conclusions should be reached

until they are constant after several examinations, with a short rest between the examinations of the several muscles. It is also advisable to test the abducting power of the externi first, as this power is as a rule more constant, and its power is also indicative of the relative power of the lateral moving muscles.

In this manner we proceed to test the power for divergence; then convergence; then sursumvergence and lastly deorsumvergence, but in all tests we must vary the procedure so as to eliminate any tendency to spasmodic action of the muscles.

Under the stimulation of the guiding sensation we find that the power of these muscles have a certain relation to each other, according to their anatomical construction and their insertion into the sclera, and I test them as follows: For the externus or abduction, we place in the trial frame a weak prism, with the base inward or toward the nose. This causes the rays of light from the object to deviate inward and fall upon the inner part of the retina, producing diplopia, at once the guiding sensation through the fusion centre will stimulate the action of the externus and the eye is turned outward until the images fuse. As they blend together we then proceed to place stronger prisms in the frame, until the action of the externus can no longer fuse the images and the strongest prism with which single vision is produced will represent the power of the externus to turn the eye outward or the extent of the field of fusion in that direction.

We then proceed to test the power of adduction in the same manner and after these results have been noted, we test the power of the eyes to turn upward and lastly downward, with very weak prisms. After these tests of all the straight muscles have been carefully made and the results noted, we will not only find a very decided difference in the power, but also that this power of each mus-

cle bears a certain relation to its opposite or antagonist muscle.

The power of the externi, in the normal eye, is usually about 6°; the power of the interni, about 20°; that of the superior, about 2°, and that of the inferior somewhat stronger than the superior, and the relative values would be represented as about three to one for the lateral moving muscles and about one and one-half to one for the vertical muscles. Now this relative power is very important, as, if they keep their relation, one to the other, I do not think it makes much difference what may be the actual power of the muscles to turn the eyes under the influence of the guiding sensation. For instance if the externi should show a power of 10°, then I would want to find the power of the interni nearly 30°, or if the externi should only show 3° then the power of the interni should not be above 10° to 12° with the vertical acting muscles in the proportion as noted above.

I have accepted this relative power, after careful examination of many cases and feel assured that when we find the relative balance, after repeated trials, as previously suggested we cannot have an insufficiency of the muscular balance.

Other observers seem to have arrived at somewhat different conclusions in reference to the standard power of these ocular muscles, but they give about the same relative power, and each one must form his own conclusions from the examinations made personally, while cases that will show a much higher degree of muscular power or a larger field of fusion, are very rare. But in all cases I believe that the most essential point is, that the relative power of these muscles should be as suggested, and if so, the eyes will be free from any muscular deficiency whatever.

If this relative power is dependent on the guiding sen-

sation we may see the importance of preserving this function in all our tests, as Savage well says, page 125, "All of the center of the oculo-motor nucleus have one common master, which is the guiding sensation, residing in the retina, its home being the *macula lutea*, and its immediate neighborhood." Hence, I have no confidence in any tests of the power or the weakness of these muscles that deprive the eye of this most important function, except so far as they may be confirmatory of the prism test as suggested and as fully demonstrated in my paper on the "Power of the Interni," read before the Ophthalmic Section of the New York Academy of Medicine, November 19, 1893.

I shall not present the arguments, for or against this method of testing the ocular muscles, that have been presented by many writers, — Noyes seems to depend on the same tests that I have advanced; also, Bannister's Monograph shows the same field of fusion. Others do not seem to claim any power or virtue in the guiding sensation, but simply base their conclusions on the tendency that the eyes have to turn in certain directions when deprived of this function. Furthermore, I would not discuss the theory that in testing one muscle in this manner we are simply estimating the power of its antagonist of the other eye, as we will meet with many cases that are by no means simple in their final results. I have endeavored to study this muscular action in the direction of the indications given to us by nature, and in my treatment to place the power in their relation to each other as obviously intended.

In this connection and for the sake of argument we will refer to Stevens, page 199, "A prism with its base down before one eye is equivalent in its action to a prism with its base up before the other," and Hansell of Philadelphia advances the same suggestion, but I cannot clearly under-

stand this theory, which implies that if I put a prism of 6° over my right eye, with the base down, so forcing the eye to turn upward under the stimulation for fusion by the guiding sensation, how then will there be any innervation of the left inferior and so force a strain on the opposing muscle to keep the image of the candle in the visual line, when there is no stimulation, in that eye, from the guiding sensation?

When I test my eyes and fix them on the candle at twenty feet and then place a prism over one eye, base up or down, the position of the candle does not move, nor tend to move, unless I voluntarily turn both eyes upward, if the base of the prism is down. I think that we must have the guiding sensation to stimulate the center for fusion, before the eye will move, even though the eyes act together in their associated movements. There may be exceptions to this suggestion.

All this can be proved, if we place Snellen's test-type behind the candle and test the visual power in each eye, at the same time. If the eye that is not covered by a prism deviates even one or two degrees, the visual power will at once be reduced, then as long as the visual power is up to the standard we know that eye must be placed so that the visual line is fixed on the object at twenty feet. If there should be any movement of the eye not covered, the guiding sensation will at once act to bring the image on the macula. I think the above proposition may be proved in nearly all cases.

In a paper read before the Clinical Society of the New York Post-Graduate School, and published in the *Post-Graduate Journal* for May, 1896, I have pointed out the indications for treatment, that we may find as we proceed to test the action of the ocular muscles in their relation to each other. The literature of this subject shows that nearly all of our prominent writers have suggested their

own methods; as Noyes seems to depend mostly on the prism test, Schiotz gives us the fusion far-point and convergence near-point; Maddox, the rod-test; Landolt, the meter-angle, while Stevens, having taught us the new nomenclature, and the diagnosis of the various conditions of heterophoria, now places his estimation not on the fusion power, but on the extent of the field of fixation, as shown by his tropometer. But can there be a standard, such as we have universally adopted in the visual power of the eyes? And as we use the glasses to test that visual power may we not use the prisms to estimate the relative power of the ocular muscles and to find any existing insufficiency, with the indications for treatment?

If so, then we shall only consider nature's work and her needs in this extremely interesting study; that the guiding sensation or fusion power of the eye is the one and only stimulation by which the binocular visual power is controlled and that nature has obviously, in her anatomical construction of the ocular muscles, pointed out to us the needs and necessities of the eyes, in their movements about the center of rotation, in reference to the fusion power. We have the field of vision, shown by the perimeter, so, similarly, we have the field of fixation, represented by the power to turn the eyes on the center of rotation, and lastly the field of fusion, represented by the power to blend the rays deviated by a prism, each smaller in extent than the other, and each in inverse proportion to the other in importance, and that the only efficient and reliable tests for the relative power of the ocular muscles are the prism and that of the tropometer. Then a careful consideration of all the indications, as shown by these tests will lead us to the intelligent and successful treatment for the restoration of the balance of the ocular muscles, either in fixed or latent squint.

It will be noted that in my explanation of the move-

ments of the eyeballs I have not considered the various planes of motion, nor that of the axes around which the eyeball may rotate by the action of the ocular muscles. These planes and axes can be studied in our text-books on this subject, and I do not consider them as of any special importance in this connection. The movement of the visual line will pass from one point to another by the shortest and quickest route, and not along one plane and then in the direction of the other — in other words, the eye will not come back to the first position and then to the required point, but simply passes from one point of regard to another, turning about any axis relating to these two points that passes through the center of rotation. But to fully understand the motions of the eyes by the four recti we must consider *two principal* axes of rotation, the vertical and the horizontal, with their respective planes of action, in reference to a full understanding of the actions of the muscles themselves. These four muscles we may consider as acting in pairs, or as one muscle antagonistic to the other. The internal and external recti will then by their most simple action move the eyeball in the horizontal plane, turning about the vertical axis, and the superior and inferior will move the eyeball in the vertical plane about the horizontal axis. These are the two simple movements of the eyeball, from which we construct the terms to express them, but as the eyeball rests in the orbit surrounded by its tissues and has no fixed attachment, then the combined action of any of these muscles will turn the eyeball to any position in the field of fixation. The action of the oblique being only concerned in torsion, will keep the two vertical planes of the eyes parallel to each other in all positions. Ellert says : "The production of motion in a normal eye is denied to the oblique muscle," and I firmly believe in that statement.

Accepting then these two axes of rotation and these planes of motion, it is essential that we should have some terms by which we can express these movements of the eyeballs in the field of fixation and in the field of fusion. The terminology suggested by Ellert, in the *Journal A. M. A.*, October 18, 1902, seems to me will meet all the requirements, and I take the liberty of giving these terms in full.

MOVEMENTS OF THE VISUAL AXIS:

A. Of a single eye (ductions):
   a. Movements of the V. A. outward = abduction.
   b. Movements of the V. A. inward = adduction.
   c. Movements of the V. A. upward = superduction.
   d. Movements of the V. A. downward = subduction.

B. Of both eyes:
   1. Associated conjunctive movements (versions):
      a. Movements of both V. A. to right = dextroversions.
      b. Movements of both V. A. to left = levoversion.
      c. Movements of both V. A. upward = superversion.
      d. Movements of both V. A. downward = subversion.
   2. Associated disjunctive movements (vergences):
      a. Inclining the V. A. toward each other = convergence.
      b. Inclining the V. A. away from each other = divergence.
      c. Inclining one V. A. higher than the other = supervergence (right and left).

SPECIAL TERMS TO DENOTE THE EXTENT OF ASSOCIATED DISJUNCTIVE MOVEMENTS AS MEASURED BY PRISMS:

a. Ability to overcome prisms bases outward = prism-convergence.
b. Ability to overcome prisms bases inward = prism-divergence.
c. Ability to overcome prisms bases downward or upward = prism-supervergence (right and left).

Ellert adds to this,

SPECIAL TERMS TO DENOTE THE EXTENT OF TORSION:

a. Movement of the vertical axis outward = extorsion.
b. Movement of the vertical axis inward = intorsion.

These terms apply to the movements of the eyeball about the sagittal axis, and depend upon the action of the oblique muscles.

Savage has suggested the term cyclophoria (plus and minus), and cyclotropia (plus and minus). I can see no reason why these conditions may not exist, as we may have a weakness of the oblique muscles, the same as that of the recti, but in the last classification if we have a malposition of the vertical plane, it must be due to a pathological condition (paralysis) when present. These cases are very rare.

In closing this discussion of the action of the muscles of the eyeball, we must not consider the eye as a rubber ball and the axes as a number of knitting needles. That is man's mechanical method, but let us study the movements of the eye and the direction of the visual line from the standpoint of nature, even if that is an "every-day affair," unfortunately not referred to in our text-books. Then we shall simply note that the recti muscles control the movement of the eyes in the field of version by their voluntary action, and in the field of duction by their involuntary action, and, lastly, that "the obliques must keep the vertical axes parallel with each other and with the median plane of the head when in the first position."

CHAPTER III.

CLASSIFICATION OF SQUINT.

It has been well established that we should have some terms to express the various conditions of squint, either latent or fixed, that may be simple and easily understood, and at the same time convey all the necessary information as to the deviation of the visual lines, or the tendency to that condition. I am more than convinced that we have no distinct dividing line between the two conditions as far as a scientific examination may reveal, and as I believe we have only one true underlying cause as the fundamental starting-point of all squint, it becomes only one of degree, from that of the slightest tendency to a deviation of the visual lines when in the first position of the eye, to that of the most extreme fixed deviation in any direction; but, objectively, there does seem to be a distinct dividing line on each side of which we may place, first, those cases in which we have a tendency to deviation — latent squint; and second, those cases in which we have a constant deviation — fixed squint.

Hence it is very appropriate that we shall have some terms that will indicate at once these two grand divisions with all their various complications, from the slightest heterophoria to that of fixed squint. In this classification I shall use the first position of the eyes as a starting-point, which, as I understand it, will be fully discussed under the condition of exophoria. From this position we may then consider not only the deviation of one visual line from that of the other, but also any deviation of both visual lines together. I do not know of a better

classification, and one that has been almost universally adopted, than that of Stevens, in which he has considered almost every possible deviation that may occur in the position of the visual line. Hence we have the following terms:

Orthophoria = parallelism of the visual lines, no tendency to deviation.
Heterophoria = the tendency to deviation from parallelism of the visual lines.
Hyperphoria = a tendency of one visual axis above the other (right or left).
Hypophoria = a tendency of one visual axis below the other (right or left).
Esophoria = a tendency of the visual axes inward.
Exophoria = a tendency of the visual axes outward.

These terms may also be combined to express any tendency of the visual axes in more than one direction, as

Hyper-esophoria = a tendency upward and inward.
Hypo-esophoria = a tendency downward and inward.
Hyper-exophoria = a tendency upward and outward.
Hypo-exophoria = a tendency downward and outward.

We have here distinct and clear expressions in a single term that will indicate any and all deviations of the visual axis from that of the first position, modified only by the degree of deviation as shown in the examination. Similarly, we have below the terms that will denote a fixed condition, simply one of degree, from that of the first series of terms:

Esotropia = deviation of the visual axes inward, or convergent squint.
Exotropia = deviation of the visual axes outward, or divergent squint.
Hypertropia = deviation of one visual axis above the other, upward squint.
Hypotropia = deviation of one visual axis below the other, downward squint.

These terms may also be combined, as

Hyper-esotropia = upward and inward.
Hyper-exotropia = upward and outward.
Hypo-esotropia = downward and inward.
Hypo-exotropia = downward and outward.

These various conditions may exist in any degree, and in fact, it seems to me that all squint of whatever variety is simply one of degree rather than one of kind, as in one case the deviation is obvious and more or less fixed, in the other the deviation can only be detected by the most careful examination by the present methods. Again, even these two conditions show many different degrees, as in fixed squint the deviation of the visual lines may be from that of the slightest to that of the greatest degree in any direction.

Stevens has also given us other expressions that will indicate the deviation of the visual lines together, as one case may have a tendency to deviation of both visual lines above the plane of regard, and another below it. Hence we have these terms:

Euphoria = 33° up and 50° down, or normal rotation.
Anaphoria = a tendency of both visual axes upward.
Kataphoria = a tendency of both visual axes downward.
Anatropia = a deviation of both visual axes upward.
Katatropia = a deviation of both visual axes downward.

To these conditions I would add certain tendencies of the visual axes to turn to the right or left, in which, by the examination, we find a weakness of one internus and one externus, giving a tendency to look to the right or left. I do not propose to suggest any special term for this condition, but as I have met some cases, prefer to note it in this classification and to explain it under the subject of Heterophoria.

Lastly, to make this classification complete, although

somewhat beyond the scope of this monograph, we have the condition in which there is a tendency to the deviation of the vertical meridian of one eye from that of the median vertical plane of the head:

Plus Cyclophoria = a tendency of the vertical axis from the median plane of the head.

Minus Cyclophoria = a tendency of the vertical axis toward the median plane of the head.

These terms are here used for the purposes of uniformity, as Stevens' designation is respectively, "Plus declination and minus declination," which seem to me much more appropriate, and may be simply considered as complications of squint. Consequently they will not be fully or separately discussed, except so far as they may complicate any deviation of the visual line.

CHAPTER IV.

ESOPHORIA, OR LATENT CONVERGENT SQUINT.

"If Esophoria be regarded as latent, then Convergent Squint may be considered as an Esophoria which has become manifest and immutable strabismus."—*Campbell.*

Ophthalmologists have not agreed upon the exact method of examination that we should pursue in the estimation of an imbalance of the ocular muscles, nor are the results of our final work the same in all cases, even though we may be much more exact now in the estimation of the refractive apparatus. This is particularly so in the estimation of the motility of the eyes, in other words the power of the eyes to move in the fields of fusion and version. The same difference of opinion seems to exist in the other branches of our profession as I believe it is not fully settled yet, whether antitoxin is a positive specific for diphtheria, and as to the right and proper time to operate in all cases of appendicitis. Medicine has not yet reached that stage — nor do I think it ever will — when we can make the condition of all our cases an exact science by which we can tell just what will be the final result. We must deal with the personal equation of our patients, an extremely varying quantity. If others may have these diverse opinions, so do the ophthalmologists hold the same in reference to the subject of the motility of the eyes. Some may believe that latent squint does not exist, or if so, that it simply requires the use of glasses; others, that nearly all cases of asthenopia present this condition and that the majority of cases will require one or more operations.

For my own views and from my experience I must say I am inclined to take a conservative course, simply

endeavoring to correct the refractive error, whatever that may be, and then to place the muscular apparatus, if necessary, in that condition of equilibrium that nature obviously intended. Having done this and the patient finds no relief I can justly feel that the eyes are not at fault. My methods of examination and conclusions may not be correct — as they have not been approved of by many other examiners — but they have at least the advantage of having been used for many years by myself with continued satisfaction.

Now there is much diversity of opinion in reference to the muscular equilibrium of the eyes. When there is an absence of this condition the writers state that the fundimental cause is hypermetropia, and then proceed to explain the relation between cause and effect, or, we may say the physiological connection between accommodation and convergence, to account for it ; but they fail to point out why the same condition may and does exist when the refraction of the eye is found to be myopic. So we find this latent tendency with myopia described in "vague terms" as ciliary spasm — a rare condition in myopia — or we find no explanation at all ; or, again, it is so described that the reader cannot decide what is the chief or primal cause of the condition and he is entirely at sea as to what procedure he shall adopt when he fails to correct and relieve his cases with the use of glasses, or by the method of exercising the muscles, so frequently suggested. It is for these reasons that I wish to offer some suggestions, or methods, based on my own experience, and by which I have almost invariably given relief.

At one time I also held the opinion that hypermetropia was the cause of esophoria and convergent squint, but, when I noticed the vast number of hyperopes that had no tendency to these conditions and that the greater the hypermetropia the less did I find this inability to keep

the visual lines fixed, then I began to doubt my former opinions. Furthermore, when I also found this condition associated with myopia and even emmetropia, I felt compelled to discard the theory of the connection between accommodation and convergence as the true cause and to look for other reasons that would meet all the indications. This can be found in the field of fusion or the desire for single vision, and in the field of version or the rotation of the eyes. The desire for fusion must be controlled by a different part of the brain from that of fixation, in other words, one is automatic and the other is control.

The power of an eye muscle to act according to its physiological function is shown, first, by the ability of the individual to do certain acts by the process of muscular contraction, stimulated by the innervation and controlled by the will-power and, second, the ability to turn the eyes, one independent of the other, under the stimulation of the desire for single or binocular vision. This latter power is the fusion force, and is not under the control of the will-power of the individual. Under these conditions we find that the eyes can turn to all parts of the field of fixation in nearly the same degrees of the arc of a circle, while, on the other hand, the eye will also move to a certain extent and within a certain field (*fusion*) not by the will-power, but by that of the muscular apparatus, under the stimulation of the center for fusion, due to the desire for single images or that the images may be on corresponding parts of the retina. This power of the muscular apparatus seems to be limited within a certain field, and we find this fusion force acting in the same way when the eyes are directed to the different parts of the field of fixation. Hence the ability to fuse these images must be controlled by a special center, and the extent to which the eyes can be turned to produce this fusion must represent the power of the muscles to turn the eyes. I can see no

other way to consider the action of the ocular muscles than their individual power to turn the eyes under certain conditions. Moreover, I cannot consider this force as innervation, nor that there should be more stimulation to one muscle than to the other, since the size and the insertion of all the straight muscles seem to point to the fact that one muscle must have more power than the other for some special purpose; or, in other words, exert a greater power to turn the eye under the stimulation of the fusion force.

The physiological act of turning the eyes in all directions within certain limits requires very little force, as is well shown by the action of the interni and the superior recti muscles, for the eyes will turn upward as well as inward, and yet the size and the insertion of the interni clearly indicate that these muscles must have been intended to exert a greater power in some way. May we not also accept the supposition that nature arranged this muscular force, not only to perform the simple act of turning the eyes, but that they might also conform to all the requirements of the more important functions, namely, that of binocular vision and convergence, as controlled by the fusion force of the eyes? Do not the daily needs of vision confirm all this? It is in the act of single vision, both for the distant and near points, that we find the eyes constantly called upon to keep the visual lines fixed on an object, while at the same time the eyes are moved to different parts of the field of fixation. Upon this supposition I cannot see what influence innervation, the position of rest, or the natural elasticity of the externi, have to do with the fusion of the images on the retina. If we accept this reasoning and believe that certain muscles have a greater power to turn the eyes under certain stimulation, then we should have some test sufficiently reliable and exact which will meet the requirements and indicate

to us what is the actual power of these muscles to turn the eyes under certain conditions. I have used and tried faithfully all the various tests as suggested for this purpose, such as the phorometer, Maddox rods, etc., but I can find no test so reliable and so clearly indicative of the power of these muscles, as the old and simple prism test. We turn the eyes in the field of fixation under the stimulation of the will, simply because we wish to see in certain directions. This is the conscious part of the action of the eye-muscle; but having performed this act we now call into action the unconscious part, in which the eyes must adjust the visual line according to the direction of the rays of light as they pass through the dioptric media. If these rays of light are deviated from their natural path the eyes will turn on the center of rotation to meet the deviated rays until the visual line will be parallel with them and the principal axial ray falls upon the macula. If I deviate the rays passing into one eye 20° from a direct line, then to have single vision there must be sufficient muscular power to turn the eye in the direction of the deviated ray, namely, 20° of the arc, and if we cannot fuse the images then we do not have sufficient muscular power in the muscle so tested. Therefore, the strongest prism that will deviate the rays and yet have single vision remain must indicate the power of that muscle to turn the eye. Now, if the deviation by a prism represents the power of the eyes to turn on the center of rotation under the stimulation of the fusion force (*duction*) what do we find is the *relative force* or power of these straight muscles? Evidently we may have some standard of comparison, in the same way and for the same reason that we have a standard for the acuity of the vision.

First, the power of the externi to turn the eyeball outward must be considered. Let me state at once that I do not consider this power as acting from the position of ex-

treme convergence or any point midway between that of extreme convergence and divergence, as has been suggested by some writers, but as starting from the usual first position of the eyes, in which they are directed toward a point about twenty feet distant and about 15° below the horizon. Second, the power of the eyes to move under the stimulation of that unconscious force or duction, as shown by the prism test, must be measured in the remaining parts of the field. We should find the outward movement or abduction about 6°. Any decided variation from this *may* indicate some latent squint. The inward movement or adduction is about 24°; the upward movement or sursumduction about 2°, and the downward movement or deorsumduction about 3°, or somewhat greater than the upward power. These movements are indicated by the relative size and insertion of the straight muscles, and very beautifully represents the power of the muscles to turn the eyes under the desire for single vision, while the slightest failure of this muscular power may result in diplopia. Now, in the above standard we shall find a certain relation in the power of these muscles, one to the other, as the interni are the most powerful; next the externi; then the inferior, and, lastly, the superior, each one having a certain ratio to the other. When this is constant — no matter what the actual power may be — we will not, as a rule, find much if any muscular asthenopia or tendency to latent squint. Having, then, this standard by which to compare the results of our tests of the muscular balance as found in our patients, what will be the indications of the condition that is the subject of this chapter? At once we note that the power of the interni is far greater than that indicated by our standard, or *vice versa*, that the power of the externi is too low, so that under the stimulation of the fusion force the power of abduction is not great enough to control the position of the eyes except by the excessive stimulation of

the externi to keep the visual lines fixed. These patients present the usual train of symptoms, chiefly pain in the head, radiating backward toward the neck, and a pulling sensation about the eyes. Latent squint of the convergent class is sometimes described as spasmodic action of the interni, and in some cases our tests show this condition; but even if it is spasmodic action of that muscle it still indicates too much power, and should and can be controlled by proper and suitable means. Some of my best cases have shown this condition, as I have noticed a *peculiar* change in the response of the externi to the prism test, in which there seems to be a decided loss of power *after* we have tested the adduction. This is shown by the prism test as well as by the phorometer or the Maddox rod. If we test the power of abduction we find a certain ability to turn the eye outward under the stimulation of the fusion force, that as measured by the prisms may be as high as 6°. Now, testing the adduction in the same way we have 20° or more, showing a fairly good balance between adduction and abduction; but if we go back and again test the power of abduction we will find that it has become reduced to 1° or 2°, or possibly we will have homonymous diplopia — particularly if we place a red glass before one eye — which may persist for some time before the balance again adjusts itself, and we have single vision. I have considered this a very valuable test to develop the tendency of the eyes to turn inward, and while it may be due to some spasmodic action of the interni, yet I consider it one of the best indications for an operation. This condition of the externi or weakness of the muscle will also be shown in the test of the field of version by the tropometer. In this field we will find the voluntary power to turn the eye outward much less than normal, while the inward rotation will probably be greater.

Having found the conditions of latent squint, conver-

gent, our method of treatment for its correction is, first, the use of suitable glasses to correct any existing refractive errors. This failing, the combination of prisms with the glasses may be tried; then possibly tenotomy of the interni; but best of all, in my opinion, is the operation for shortening the externus with a catgut suture, thereby increasing its power to turn the visual line outward and at the same time avoiding all danger of an overcorrection. It is to show the results of this operation of shortening the ocular muscles for the correction of this condition that I present these cases from my private case-book, and also to demonstrate the utility of this standard test:

CASE 1484.—Hy. with Ah. glasses four years. V=20/20, Ad. 20°, Ab. 2° (ratio 1 to 10). Operation: Shortening of Ext. Rect. Result, Ad. 15°, Ab. 4° (ratio 1 to 4).

CASE 1755.—Hy. with Ah. glasses several years; no relief. V=20/15, Ad. 25°, Ab. 2°; after testing Interni, Ab. 0. Was told by another oculist that operation would do no good. Operation was shortening of Left Externus. Result, Ad. 15°, Ab. 5°. Two years later reports can use the eyes all that is needed with comfort.

CASE 1829.—Ah. glasses. V=20/15, Ad. 30°, Ab. 4°. Operation: Shortening of Left Ext. Rect. One year after is so much improved that she returns and asks for an operation on the other eye, and I shortened the Right Ext. Rect. in the same way, with this final result, Ad. 20°, Ab. 5°. Can now use the eyes with comfort.

CASE 1839.—Hy. with Ah., using glasses; no improvement. V. 20/15, Ad. 15°, Ab. 0°. Operation: Shortening of Left Ext. Rect. Result, Ad. 16°, Ab. 3°.

CASE 1909.—My. with Am., using glasses. V=20/15, Ad. 20°, Ab. 4°. After testing Interni, Ab. 0°. Operation: Shortening of Left Ext. Rect. Final result, Ad. 15°, Ab. 6°. Two years after reports no pain or asthenopia.

CASE 1942.—Hy. with Ah., using glasses. V=20/20, Ad. 10°, Ab. 1°. Operation: Shortening Left Ext. Rect. One year after has Ad. 12°, Ab. 4°.

CASE 1977.—Hy., using glasses. V=20/15, Ad. 25°, Ab. 0°. Operation: Left Ext. Rect. Final result, Ad. 20°, Ab. 6°.

CASE 2051.—Ah., using glasses. V=20/15, Ad. 30°, Ab. 6°.

After testing Interni, Ab. falls to 0°. Operation on Ext. Rect. Final result, Ad. 20°, Ab. 6°, six months after operation.

CASE 2065.—My., using glasses. V=20/15, Ad. 30°, Ab. 3°. Operation, Ext. Rect. Final result, Ad. 24°, Ad. 6°.

CASE 2292.—Hy. with Ah. using glasses. O. D.=20/20, O. S.=20/40, Ad. 30°, Ab. 3°. Operation. Final result, Ad. 20°, Ab. 6°.

CASE 2371.—Hy. with Ah., using glasses. V=20/20, Ad. 25°, Ab. 6°. After testing Interni, Ab. 0°. Operation: Shortening Right Ext. Rect. One year after, Ad. 25°, Ab. 6°, permanent.

CASE 2385.—Ah. ax. 180°, using glasses. V=20/15, Ad. 20°, Ab. 4°. After testing Interni, Ab. 0°. Operation: Shortening. Four months after, Ad. 15°, Ab. 4°.

CASE 2088.—Ah., using glasses. V=20/15. Better with glasses, but not relieved. Ad. 20°, Ab. 1°, and homonymous diplopia after testing Interni. Operation: Shortening; Ab. 4°., much better.

CASE 2459.—Ah., using glasses. V=20/15, Ad. 25°, Ab. 2°, falls to 0°, with homonymous diplopia after testing Interni. Operation: Shortening. Six months after, Ad. 15°, Ab. 5°.

CASE 2621.—Ah. V=20/15. Glasses do not stop asthenopia. Ad. 20°, Ab. 1°. Operation: Shortening. Result, Ad. 15°, Ab. 4°.

CASE 2571.—Ah. ax. 180°. V=20/15. Glasses relieve at first, then fail. Ad. 15°, Ab. 1°; homonymous diplopia with red glass. Operation: Shortening. Result, Ad. 15°, Ab. 5°; complete relief.

These cases usually present the history that the patients cannot read; the eyes are painful; headaches, frontal and usually extending backward, are experienced, and there may be car-sickness, nausea, etc. I have presented the histories of these cases of esophoria, or latent convergent squint, in the simplest condensed form possible, noting the refraction, glasses worn without relief, acuity of vision, the muscle imbalance, the muscle operated upon, and the final result. They all seem to show a want of power in the external recti muscles, a tendency for the eyes to turn inward, and, finally, show an improvement in that power by the muscle balance after the operation. Now, I would state that the same operation was performed in each case, namely, that of shortening the muscle by the insertion of the catgut suture by a simple method so as

to form a "tuck" at the insertion of the tendon into the sclera, and allowing the suture to be absorbed. It is fully detailed in the article on operations.

CONDENSED HISTORIES OF CASES OF ESOPHORIA.

No.	Vision; Age; Rt.	Vision; Age; Lt.		Refrac.	Add. first	Abd. ex.	Tropomet. in out	Operation, tuck. ext.	Operation, ten. int.	Esoph. deg. before oper	Add. last	Abd. ex.	Remarks and after effects
1	15	15	50	Ah.	6°	6°			both		20°	8°,	
2	20	20	35	Ah.					both	8° to 25	15°	5°,	almost squint
3	15	15	36	My.wAm.	50°	1°		L.		4°	25°	6°,	good 9 years
4	15	15	30	Hy.	25°	2°		L.		4°	20°	3°,	
5	20	20	17	Am.	20°	6°		Tuck &	Ten. R. Sup.	4° Hy. 4	20°	6°,	
6	20	20	20	Am.	10°	1°		R.		2°	10°	3°,	exer. no use
7	20	30	38	Ah.	12°	0°	55°–40°	L.	& L. Sup.	6°	12°	4°,	had prisms
8	15	15	26	Ah.	25°	1°	50°–40°	R.	Reports	no imp.	in symp.		
9	15	15	31	Hy.wAh.	15°	3°	50°–45°	R.		2°	15°	4°,	50°—50°
10	15	15	41	Ah.	15°	2°	50°–40°	L.			20°	5°,	45°—45°
11	20	20	33	My.wAm.	60	6°	55°–45°	L.		6°	50°	15°,	good 1 year
12	15	15	43	Ah.	15°	2°	50°–40°	L.	& L. Sup.	3°	15°	5°,	45°—45°
13	15	15	34	Ah.	12°	2°	50°–40°	R.		3°	12°	4°,	40°—40°
14	15	15	24	Hy.wAh.			50°–25°	L.	L.	10°			50°—35°
15	15	15	45	Hy.	15°	4°	50°–40°	L.	L.	2°	15°	5°,	40°—40°
16	15	15	43	Hy.	20°	3°	45°–35°	L.	L.	5°	16°	6°,	40°—40°
17	20	20	28	Hy.	15	1°		L.		6°	20°	6°,	
18	15	15	20	Ah.				L.		10°			good 2 years
19	15	15	18	Ah.	10°	2°		R.			10°	3°,	
20	15	15	25	Ah.	40°	4°			both	4°	35°	8°,	
21	15	15	38	Ah.	30°	4°		L.		5°	20°	5°,	good 4 years
22	15	15	40	Hy.	25°	2°		R.		3°	15°	6°,	45°—45°
23	20	20	23	Hy.	15°	0°		L.		4°	15°	3°,	
24	20	20	36	Hy.wAh.	12°	1°		L.			12°	4°,	
25	40	200	29	My. & Hy.	20°	1°	Hyper. 1°	L.		8°	20°	5°,	
26	20	20	51	Ah.	30°	4°		both			20°	5°,	
27	20	20	24	Hy.	15°	1°	Hyper. 4°	L.		4°	20°	6°,	
28	20	15	35	My.wAm.	20°	0°		L.			15°	8°,	
29	15	15	32	Ah.	30°	2°		R.		10°	28°	8°,	
30	15	15	47	My.	30°	4°		L.			24°	6°,	
31	15	15	20	Ah.	20°	2°	Hyper. 2°	L.		3°	15°	4°,	
32	15	15	20	Ah.	20°	0°		L.			15°	3°,	
33	40	20	24	Ah.	25°	2°	Hyper. 3°		L.	4°	25°	8°,	
34	15	15	34	Ah.	15°	1°		L.		6°	12°	3°,	Petit. Mal.
35	15	15	28	Ah.	25°	1°		L.		6°	15°	5°,	
36	20	15	34	Hy. wAh.	20°	1°		R.			20°	4°,	
37	15	20	47	Ah. & Am.	12°	1°		L.			15°	5°,	
38	20	40	40	Ah. & Am.	15	1°		L.			20°	5°,	Petit. Mal.

Explanation: Visual acuity, the numbers are the denominator with 20 as the numerator in all cases. Prisms were used in some cases but failed. Results: In all these cases there was a decided improvement in the asthenopic symptoms except in the one case noted. All of them had worn glasses some time with the most careful correction of the refraction, but with no relief. Orthoptic training was tried but did not give any results. Thirty-four operations for shortening the externus were performed, and in a few a partial tenotomy was combined with it. Six cases showed some hyperphoria. The measurements in the field of version all show improvement. The asthenopia in the cases of Petit. Mal. was improved.

In closing let me say I am trying to follow nature in the movements of the eye, and, to quote from Professor Tyndale: "In dealing with nature the mind must be on the alert to seize all her conditions; otherwise we soon learn that our thoughts are not in accord with her facts."

CHAPTER V.

EXOPHORIA, OR LATENT DIVERGENT SQUINT.

This condition presents certain peculiarities that are not met with in its opposite, esophoria. Its correction when glasses are used seems almost impossible; successful results of our operations seem more difficult to obtain, and the etiology, primarily, of this tendency to divergence seems so difficult of solution that any testimony that will elucidate this subject is desirable, while any theory that may include all cases, not paralytic in their nature, may be acceptable to the profession, though based solely on the results of one's own limited experience in this field of ophthalmology. I may then state that if I express a suggestion or condition that will give some one sufficient data for reflection I shall be more than repaid for this effort. The question of what is exophoria is simple enough; we all know the term implies a tendency to divergence of the visual lines, or as some of my colleagues prefer to call it, "insufficiency of convergence," or "excess of divergence," but the nature and etiology of this tendency of the visual lines to diverge is to me a question of great importance. We cannot advance a suggestion as to the etiology unless our explanation may and does cover *each and every case*, primarily, though many other causes may be contributing ones. I do not feel satisfied that we should say this case is due to an insufficiency of one function, or an excess of another, nor that an excess of, or want of, innervation of the ocular muscles is the cause of this divergence, but I am more and more convinced that it must be in the anatomical construction and insertion of the ocular muscles, probably congenital; in other words, we must look for the

cause primarily in the muscles themselves, and principally in that of the interni. All the various theories cannot be correct, so the question arises: What theory is the best? And is the cause a theory only, or a demonstrable fact?

* "The conception of exophoria that seems to be nearest to physiological truth is that the outward tendency of the visual axes results from the loss of convergence-impulse and hence must be considered as a passive rather than an active condition." If this is a physiological truth then there must be some fault with the examination of the musculature of the eye at the present time, as it does not agree with my own examinations and experience, and hence the reason, in part, for offering this work to the profession. The movements of the eyes in the orbits are truly wonderful physiological phenomena, partly under the control of the will, voluntary; partly under the control of the center of convergence, whose limits extend from the first position of the eyes to that of the most extreme convergence, and partly beyond the will-power, involuntary. These functional conditions of the eyes have been so adjusted by nature that, in my opinion, there is established a natural and a normal tonicity of the ocular muscles which places the eye in a position of rest when controlled by the visual power, and also, † "because the eyes naturally tend to remain in this first position of the eyes, as even in cases of complete paralysis of all the lateral moving muscles of the eyes the look is in infinity." This position, which we designate as the first position of the eyes, is one from which they may be moved, by the *voluntary* action of the will, to any secondary position within the limits of the field of version, consensual and also convergent to

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* Hansell & Reber. Mus. Anom. of the Eyes, p. 100.

† See Archives, March, 1903. Wernicke on Paralysis of Latero-version.

the near-point and by *involuntary* action, within the limits of the field of fusion. * Maddox evidently seems to consider the position of rest as that of the first position; in other words, the position of the visual lines when the look is in infinity and slightly below the horizon, yet he does not consider adduction and abduction as starting from this point. Furthermore, it is an evident fact that convergence only extends or is controlled from this position to that of the most extreme near-point, and if so can have no action, either active or passive, to produce the condition of exophoria, a tendency to deviation from this first position.

According to Savage he would place † "the primary position of an eye in which the visual axis is in a fixed horizontal plane of the head and at the same time parallel with the median plane of the head," but this position in the normal states, as he well says, "can never be," except in that unnatural condition, "narcosis, or death." The first position of the eyes must be found, not by any theoretical arguments, but from the position as ordained by nature, for "Nature is truth." The statement that divergence is a normal condition in which the eyes seek the position of rest cannot be verified by any actual experience that I am familiar with; on the other hand, my investigations have led me away from such conclusions, and that the position of rest is that of the first position, in which there is a normal balance of the ocular muscles, and that if otherwise it is due to some anomaly in the ocular muscles involved.

‡ Hansen Grut well says, "All strabismus theories, Schering's, Stilling's, Whalfor's and my own, for instance, are unsubstantial of course," but let us remember that behind all the movements of the eye we have a human being that

* Med. Jour., April 1, 1893.
† Oph. Myology.
‡ Prize Essay.

must and will use the eyes as nature intended. If we read her indications aright we will understand what is intended as the perfect standard and then we can better appreciate those anomalies so often seen in nature's works, not only in the eye but in other parts of the human system. Now Hansen Grut and also Knapp tell us that the position of rest for the eyes is one of divergence, as to quote the former: * "This position of rest for the eyes is one of divergence, rarely parallel, and hardly ever convergent," and bases that opinion on "the form of the orbit, the insertion of the optic nerve, and the natural length of the muscles when not innervated," but even if these anatomical conditions do exist what have they to do with the position of rest for the eyes? Divergence is said to be the natural position of rest, as shown when the eyes are free from innervation as in narcosis or death, but as Sweigger well replies, † "Narcosis and death are not natural conditions," and in the living conscious human being we have only to do with the functional position of rest and the standard by which we compare the various anomalies that may occur. Baker, writing on the Anatomy of the Eyeballs, says: ‡ "During sleep or unconsciousness the eyes turn slightly upwards and inwards," but this does not agree with the observations of others nor my own as I do not think that during sleep, unconsciousness, or death, the eyes occupy any one special position, as they are deprived of their most important function, the guiding sensation or fusion power. I have given some attention to this subject and had one of our staff at the New York Post-Graduate Hospital make some records for me as to the position of the optic axes during complete anesthesia, and they were not found to be in any one direction. I give the report in full, taken by Dr. H. A. Houghton who had charge of the anesthetic during all the operations

* Price-Essay. † Archives. ‡ N. & O. System.

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"Observations in a series of forty-three cases in adults, post-operative, while still under ether anesthesia, on relative position of the optic axes:

Normally convergent	11
Slightly divergent	11
Markedly divergent and upturned	6
Right, slightly; left, normal	5
Left, slightly divergent; right, more convergent	2
Right, divergent, and left convergent	4
Left, divergent; right, normal	2
Both slighly upturned and normally convergent	2
	43

Further observations seem to indicate:

(1) All of the cases classed as "markedly divergent and upturned" were in patients of very low vitality, undergoing severe abdominal operations and in a state of shock.

(2) Babies rarely exhibited any other than normal convergence while under ether. (Not included in the above).

(3) Relative positions of the optic axes varied much during operations, and in many cases observations would hold true only at the time they were made.

(4) It could hardly be said that any single patieint preserved the same depth of anesthesia throughout the whole operation. The degree of course varied with the work being done at the time by the operator.

(5) Patients came to the table with various medications such as strychnia, morphia, etc., and also received the medications while on the table.

(6) Pupillary reaction showed the same diversity as that exhibited by the optic axes.

From these excellent observations and my own clinical experience I think that the visual lines have no special direction when deprived of their physiological function and that, when the mind is in an active state, the natural position of rest is with the visual lines directed forward toward the horizon to a point slightly below it and slightly convergent. Primitive man always uses the eyes in the position of rest as he looks in the distance and, as is well

known, his vision is far more acute than that of those persons who live an urban life, and pass the majority of the time within the walls of their homes.

Hence we will here find our standard position of rest, our natural position of the visual lines, and from this position we may understand the various anomalies of the eyes as we meet them in the consultation room. Now in the study of any tendency of a deviation of the visual lines we must start from this natural position of rest, and in the subject presented we have a tendency of the visual lines to diverge from that position. It goes without saying, that is exophoria, or latent divergent squint. Then why do the eyes tend to this abnormal position, when we have found the standard or true position in which they should remain without any conscious effort or innervation on our part? Surely not because the innervation of the externi is too great, or that of the interni less than normal. Glasser says: * "We cannot have a continuity of function without a physical organization through which it functionates, and no continuity of life or feeling without continuity of a regular and systemic physical organization of the body that binds it into this systemic and related unity." And again, Reber says: † "When some member has discovered and made practical the use of a myoplegic that will paralyze the extraocular muscles as a mydriatic does the intraocular muscle, we shall have made giant strides toward solving the physiology of the extraocular muscles" . . . "It would probably upset some of our pet notions of today and require us to build up new standards." Perhaps this paper may suggest a method to show just this action.

Stevens makes the statement that exophoria is an anomaly of the ocular muscles much less frequent than

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* Med. Rec., Oct. 1902.

† Jour. Am. Med. Ass'n, Jan. 1901.

esophoria, and he concedes that it may be a congenital condition in a certain proportion of cases, but why not in all to a certain extent? Stevens does not offer any very strong evidence to the contrary. Landolt's fourth proposition seems to me to offer a suggestion that meets with my own views on this subject, in which he says: * "Finally, the fourth form of insufficiency of convergence is represented by cases in which the internal recti muscles are really weak (or, I would add, the externi too strong), and in which the field of fixation shows a limitation at the nasal side. It is the muscular insufficiency which gives rise to muscular asthenopia when binocular vision exists but to divergent strabismus when it does not exist." Furthermore, he says; "The existence of muscular insufficiency has been denied as if the ocular muscles alone among the muscles of the human body could neither be nor become insufficient for the work which they have to accomplish." . . . "If, instead of making theoretical systems, trouble were taken to thoroughly examine patients, notably their field of fixation (version), such statements would not be made," and again he says: "But is there not also a primary muscular insufficiency, due to a congenital defect in the development of one or even a group of ocular muscles?" Let us, therefore, leave out from our consideration all cases of latent or fixed squint, due to central causes, to neurasthenia, or lack of exercise, and admit as the direct, primary cause of exophoria a defect or want of development in the interni, or an excessive development of the externi which in all cases is probably congenital. This is repeatedly shown by the actual appearance of the tendons and muscles during operations, and by the measurements in the field of version, and in this direction we shall arrive at the true cause of exophoria, or a tendency of the eyes to diverge

* N. & O. System, p. 138, Vol. IV.

either during the waking hours or during sleep, narcosis, or death. Before proceeding further with the etiology of exophoria let me show the testimony of the innervation theory, that seems to have many adherents, but in which I can find little evidence of fact beyond the mere supposition and the statements of the writers.

Hansen Grut says: * "An abnormal innervation which brings about muscular contraction, not in accordance with binocular vision," and in divergent squint, he says: "A relaxation of convergence innervation to the position of anatomical rest." Furthermore, Hubbell says: † "A disturbance of oculo-motor innervation, accompanied by a perceptible deviation in any direction of the visual axis of one eye from the point of fixation of the visual line of the other eye." While Edward Jackson says: ‡ "How will an operation influence the innervation of the muscles acting on the eyeball, or will it leave the innervation quite unaltered?" Jackson does not answer it, and it is one of the questions that I would like to have explained, not asserted. I cannot find an answer to it by any of the writers on the innervation theory, but we do know that an operation must alter the anatomical conditions of the muscles, increasing their functions, if we shorten the muscular length, and decreasing their functions if we set the insertion of the tendon farther back on the eyeball by a tenotomy; if so, it must be very obvious that the same degree of innervation will have an increased or a decreased action, respectively, on the movements of the eyeball in the field of fusion and in that of fixation. Furthermore, can this innervation theory be proved by any actual experience? If we study the movements of the eyeballs in the field of version, the

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* Prize Essay.

† N. Y. State Jour. of Med., Nov. 1901.

‡ Med. News, Nov. 1902.

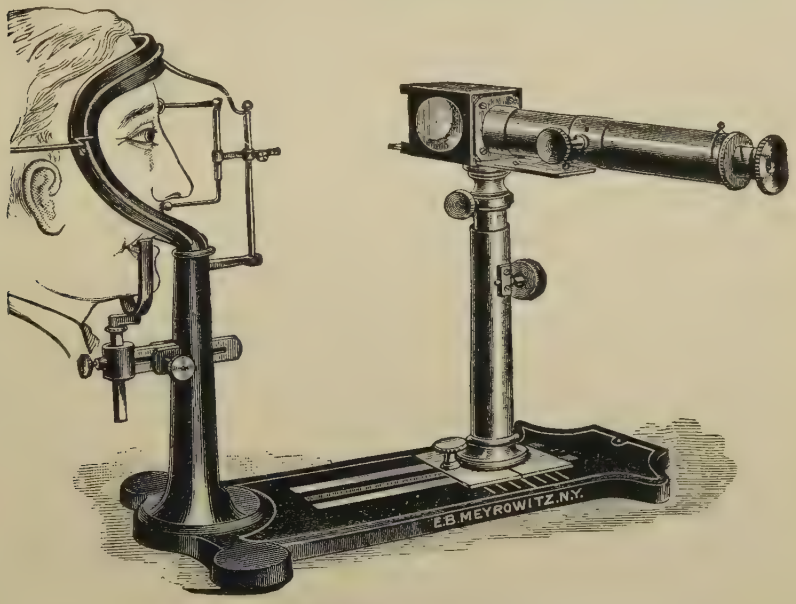
extent of that field must be limited by the actual power of the muscle to turn the visual line, during contraction, to the greatest extent of the field. Here is all the innervation and all the muscular power. Does an operation change the innervation or the anatomical conditions? The answer seems obvious. In many cases the turning of the visual line is far below the normal, though there is no diplopia, and the patient may exert his greatest will power. This seems to me due more to the anatomical construction of the muscles and to their insertion than to the degree of innervation.

In the movements of the eyeballs in the field of version there is little or no resistance, except that of the antagonistic eye muscles, which we know are relaxed to meet all the requirements of the contracting muscles, hence, innervation can and does influence the movements of the eye as the needs of vision require and the actual power of the muscles will allow to the extent of the field of version. When we consider the statement that exophoria is due or caused by myopia and the position of rest as suggested by Donders' "Antithesis," and in Hansen Grut's essay on strabismus we find some reason for these statements if we look at the question from the myopic standpoint; that is to say, if we consider the cases of myopia first, and then the imbalance of the ocular muscles. In this way we will notice that most of the cases of myopia do have a tendency to exophoria, but on the other hand, or conversely, if we study our cases from the exophoric standpoint, that is, to note the exophoria first, in all cases of asthenopia, either refractive or muscular, we find that the large majority of them are not myopic, but that hyperopia and hyperopic astigmatism predominate in about the same degree as in all other conditions of refraction or muscular imbalance. I have the records, more or less complete, of sixty-eight

cases of exophoria and thirty-seven cases of exotropia, a total of one hundred and five cases. These examinations were nearly all made in my office and are fairly correct in all the objective and subjective tests. Of this series of cases that showed exophoria we have fifty cases of hyperopia and of hyperopic astigmatism; fourteen cases of myopia and myopic astigmatism and four with emmetropic refraction, and in the cases of exotropia or actual turning of the visual lines outward, we find twenty-four cases of hyperopia and hyperopic astigmatism; seven of myopia and myopic astigmatism; one noted as emmetropic, and four in which the refraction was not noted; these latter were clinical cases. If these records are correct, and I think I can vouch for all the office examinations, then myopia and the position of rest cannot be the original cause of actual divergence or a tendency to divergence of the visual lines. Is it not reasonable, from a history of these cases, to believe we must look for some other cause for this tendency of the visual lines than the old classical ones? All these cases, by a most careful and repeated examination did show a tendency of the visual lines to deviate outward while the refraction was as carefully estimated by the objective and subjective tests, yet the cases of myopia were very small in number as compared with the total.

If this argument has any force whatever then that of the "relaxation of convergence innervation," etc., cannot be much in evidence. We have tried to prove that the position of rest must be assumed to be that of the first position of the eyes, in which the ocular muscles are in a state of normal equilibrium produced by the normal anatomical size, insertion and tonicity of these muscles. Then, if there is an anomaly of this natural condition, the eyes will tend to that position brought about by the abnormal condition of the ocular muscles. That is to

say, if the anatomical conditions of the abductors or externi will exert greater power on the lateral rotation of the eyeball than that of the adductors or interni then the position of rest will be one of divergence and only in that way. Hence myopia cannot be the determining cause of exophoria. Swan Burnett says of convergent squint, "this condition is the legitimate result of the greater power and activity of those (interni) muscles," a remark that must also apply with equal force to that of



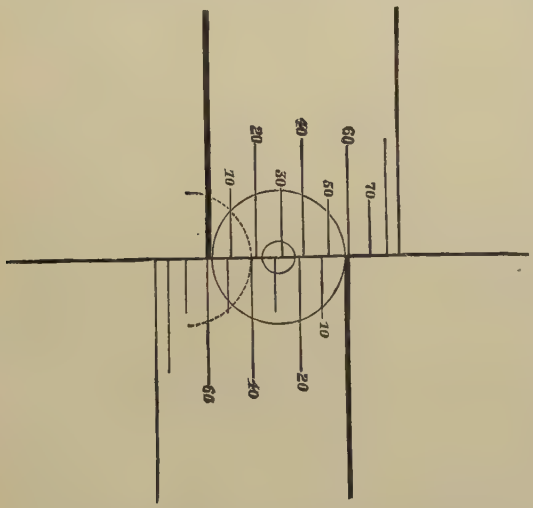
TROPOMETER OF STEVENS.

the externi in exophoria. To continue our argument as to what is the etiology of exophoria, a still further confirmation of my views may be found in the field of version. In other articles on the subject of heterophoria of the convergent class I have considered the theories of other writers and have advanced as my own that I believed it was primarily due to the muscular condition of the eyes, in other words, the power of the straight muscles to move the eyes in the field of version and in

the field of fusion. This has been called the Anatomical or Muscular theory and in my opinion is the *true* cause of all cases of heterophoria or heterotropia. I have been still further convinced of this muscular theory as the true cause of latent or fixed squint from the use of one of the most reliable instruments in the objective study of heterophoria. I refer to the tropometer devised by Stevens. This instrument measures the rotation of the eyeball about its common center in the horizontal and vertical axes by a practical and useful method, *cito*, *tuto*, *et jucunde*. It is far more exact than other methods for the measurments of the field of version, as that of the perimeter, the strabisometer, or that of noting the movements of the eye as it follows a pencil, etc. *In my opinion, it is to heterophoria what atropine is to the correct examination of refraction.* I do not think innervation can have anything to do with the movements of the eyeballs in the field of version except as they pass from point to point under the influence of the will. Does not innervation stimulate the muscles to action and do not the muscles act just so far as their individual power will permit, or, more clearly, just as far as the muscle fibres will or can contract? On contraction of these muscle fibres the eyeball may be moved a certain distance in one individual and more or less in another; one may do a certain piece of work with ease, another cannot accomplish as much, yet each may exert the same nerve force or the same muscle innervation, but one has a greater development of the muscular system, the other less. Similarly is it not so with the muscles which move the eyeballs? Nature's effort is to build us all in the same way. She has her rules that in the vast majority of individuals are carried out successfully and one muscle is always made stronger than another one, according to our physical needs, but contrariwise, in some individuals, nature seems to have

worked at cross purposes and we find a deficiency of power in muscles that should be stronger, to attain the natural uses of the system.

To illustrate, we know that in the normal field of version, as repeated in our text-books and shown in our clinical work, that the contraction of the interni is greater than that of the externi, as fifty-five is to fifty. Furthermore, in the field of fusion, the power or dynamics of the interni is greater than the power of the externi, generally as three is to one. (This proportion is generally conceded). Such is the way nature built and intended that the lateral balance of the ocular muscles should act, a fact that is well proved in the needs of our daily life. In exophoria the natural process is reversed and we find the power of the externi much greater, more or less, than what it should be normally. Hence, undoubtedly we must have the tendency of the eyes or the visual line to deviate outward. Now to resume, if we would measure the action of the lateral muscles, the findings of the tropometer being objective, it must indicate if the muscles are acting normally or if we have present an anomaly in their action. Then what is the normal action of the lateral muscles of the eyes as shown by the tropometer? Vertically, we find about eighty degrees of rotation, starting from the first position of the visual



SCALE FOR THE TROPOMETER. DOTTED LINE SHOWS POSITION OF EDGE OF CORNEA IN ROTATION.

lines, about thirty degrees upward and fifty degrees downward. Similarly, measuring the lateral movements and starting from the same position, we have about fifty-five degrees inward and fifty degrees outward. This is, in my experience, about the normal movement of the visual lines from that of the position of rest to the extreme limit of the field of version, in other words, the excursions of the cornea about the center of rotation when the eyes are normally balanced. Stevens, I believe, gives the conditions essential for this examination to be the immobility of the head; the adjustment of the instrument; the exact focus and the effort of the examiner to bring out the complete movements of the eyes in the field of version. Accepting the above measurements as that of the standard movements in this field then what will the tropometer show in a case of exophoria? I will illustrate by one of my cases:

No. 3274.—R. E., up 30°, down 60°, in 40°, out 50°.
L. E., up 30°, down 60°, in 50°, out 60°.

I think this will speak for itself as to the tendency of the eyes to turn outward, while that of the lower field seems to be a little more than the normal. In the field of fusion the eyes should turn inward two or three times greater than the outward movement and a little more downward than upward, the upward rotation being very slight if normal. As if adduction shows an in-turning, at the first examination, under the stimulation for fusion, of twenty degrees and eight degrees out-turning we will have a normal balance in that field, while any decided difference in this proportion will probably indicate an anomaly in the balance of the ocular muscles. I would illustrate this with case No. 3206, in which, after repeated trials, the abduction was 15°, and the adduction also 15°, and this disproportion between the normal and that of the excessive power of the externi is particularly obvious and shows

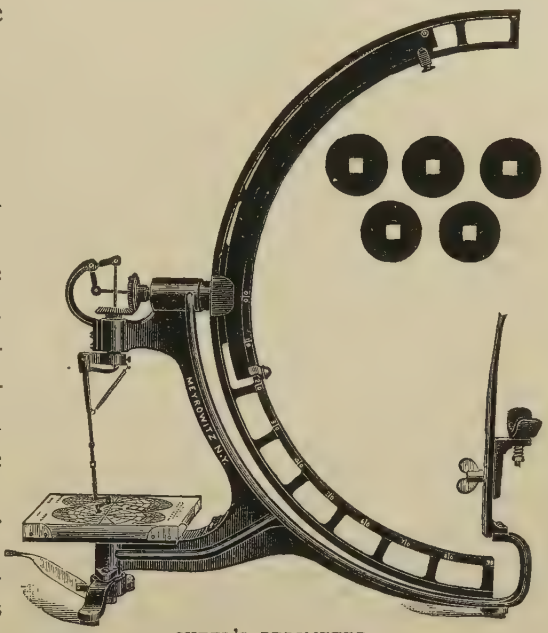
an exophoria or tendency to turn outward of the visual lines and, as the examinations in this case were repeatedly made with the same final result, the indications for its correction are easily understood.

The symptoms of exophoria may occur at any age. The time at which they complain is not fixed, that is to say, at no special time of life are the symptoms of ocular muscle strain developed. According to my records the youngest is fifteen years old, and from that up to fifty-five they have presented the evidences, both objective and subjective, of a want of muscular balance, as shown by the tropometer and the test with prisms. Their subjective symptoms are usually some of the following: Pain in the eyes, frontal headache, pain extending from the eyes over the head or back of the neck and down the spine, inability to read, nausea and vomiting, particularly when riding in the cars, localized pain in the head, lacrymation, eyes feel tired and strained, vision blurred and conjunctival irritation. Many of these symptoms are relieved by the use of glasses. The patients feel much better but still cannot continue their daily work with comfort and ease. All of my patients had been wearing glasses to full correction before they came to me and if their glasses were not correct in my estimation I invariably gave them a careful examination and full correction and these glasses were worn a reasonable time before any other procedure was attempted. As to the use of prisms, combined with the glasses, they have seemed useful in some cases, but I have little confidence in the use of prisms for constant wear and think it is much better to operate in all these cases. If they refuse to submit to an operation then the wearing of prisms may be useful for a time but my experience has not been particularly happy with their use in my office work. Very many of these cases of exophoria present the condition known as insufficiency of the interni

that is to say the power of the interni to rotate the eyes inward and to cause the visual lines to converge seems to be much weaker than normal, while the power of the externi to produce divergence is about the same as normal. In these cases I have found some benefit in a careful systematic exercise of the interni, but I consider all these suggestions, as the wearing of prisms or that of exercise of the interni, as simply putting off a procedure that should be done, as all these artificial aids eventually fail to have their beneficial effect. Of all my series of cases as reported, every one showed well marked evidence of exophoria after repeated trials of the muscular balance and all of them submitted to the necessary operations. In no case was this done more than four times and in the large majority only one or two operations were performed. The refraction of all these cases presented the usual proportion, as found in office work at the present time. The largest number being hyperopic with astigmatism and a very small number with myopic astigmatism, only one-fifth of the entire number. Of the operations performed sixty-eight were tenotomies of the externi, and eighteen, shortening of the internal rectus.

Diagnosis. The diagnosis of this condition of imbalance of the ocular muscles should not be very difficult if we accept one cause which underlies all others and some certain standard measurements that will represent the normal movements of the eyeballs in the fields of version and that of fusion. Deviations from these standards must be taken into consideration, most careful examinations made, and, unless postively indicated, all other means of relief should be considered before any operation is performed. The field of version or fixation has been well demonstrated in our recent text-books. Landolt speaks fully on these important excursions of the eyeballs taken from the arc of the perimeter and using the finest type or

two small dots as the test. One of these should be carried along the arc of the perimeter, placed horizontally to the most extreme point of clear vision, that is to say, with the head fixed and the eyes turned inward or outward as far as possible to the point where the finest type or the two dots can be distinctly seen. The degrees shown on the arc of the perimeter are the angular deviation from the first position, to the position of the visual lines in the extreme field of fixation. Landolt's chart of the normal field shows these limits of rotation of the eyeballs, though his measurements are somewhat smaller than my own or those shown by the tropometer. In the measurements of the power of each individual muscle to rotate the eyeball about the center of rotation the tropometer gives very accurate information. We do not depend upon what the patient sees, we simply note the excursions of the cornea in any direction, and measure the movements of its edge on the scale of the instrument. In considering the results of these examinations, Stevens, at one time advanced the proposition that the upward movements were the most important and the possible clue to all the conditions of heterophoria. I regret that at the present time he seems to



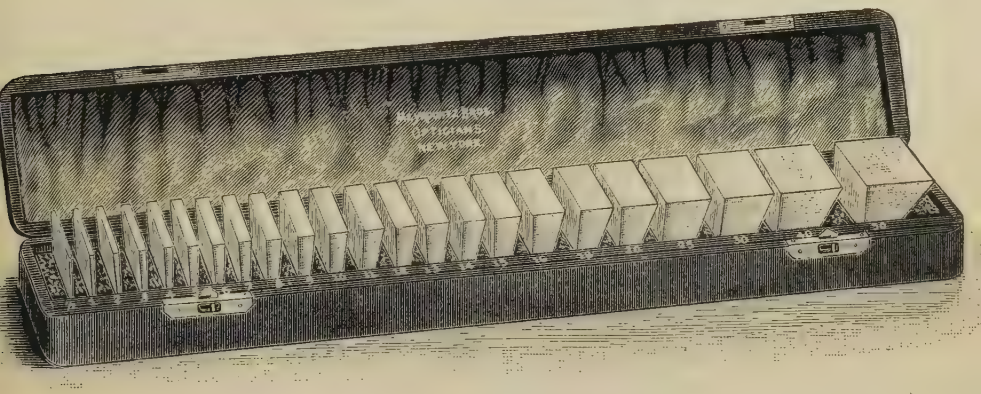
SKEEL'S PERIMETER.

have abandoned that proposition and now considers the torsion of the eyeball and its anomalies as the determining cause, yet he has not fully decided just where and when we shall find the true cause of all our cases of heterophoria and heterotropia. Be all that as it may, I still feel that while innervation may stimulate the muscles to act and that other causes may contribute to this tendency of the visual lines to diverge, yet all these conditions must be controlled by the physical power of each individual muscle to contract under the nerve stimulation and that all these cases of exophoria have a want of muscular power in the interni, which is readily and objectively shown by the measurements of the tropometer. Hence, if we accept a standard inward and outward lateral rotation of the eye and that the tropometer shows the outward rotation greater, or the inward rotation less, than normal we must have the condition known as exophoria. I accept then a normal field of version and any decided deviation from this must be due to some want of physical power in the muscles themselves. We may have cases which show a restricted movement in all directions with about the same relative degree in each. These I consider simply cases of a general anæmic condition and not those of heterophoria, but when we get a decided overaction or weakness of any one or more muscles as shown by the tropometer then I think we have some positive signs of heterophoria. To illustrate this in connection with our paper the tropometer will show :

Case No. 3206.— Exophoria. Adduction = 15°; Abduction = 15°. Tropometer, up, 25°; down, 55°; inward, 45°; outward, 50°. Or Case No. 3274.— Slight Divergent Squint. Tropometer shows, inward, 40°; outward, 50°.

Here we clearly see the difference between the rotary power of the lateral moving muscles, in which the excursions of the eyeballs are just reversed from that of the

normal relations; in simple words, greater power to turn out than to turn in. An instrument so useful and objective in its findings should be always used before any attempt is made to correct an heterophoria of any degree.



CASE OF SQUARE PRISMS.

Next in importance is that of the prism test, one that is mostly objective and very reliable. I have advocated the use of this test for many years and still feel the fullest confidence in its findings when properly used, though many of my colleagues have tried to convince me that we cannot test the power of an individual muscle in this way and that the power of one is only an indication of the power of some other muscle holding the eye in the first position, but the strongest and most important function of the eye and the keynote of binocular vision is that of the fusion power or guiding sensation. This function is not stimulated unless the rays fall upon some other part of the retina outside of the macula region. The use of this test by repeated examinations and its results confirmed by other tests will make the diagnosis of exophoria clear and at the same time indicate the most suitable procedure.

In the diagnosis of exophoria or any condition of heterophoria, I cannot place any reliance on the tests for a deficiency of power or overaction of the ocular muscles that

will deprive the eyes of their visual power in any way. That is to say, we must not lower the visual acuity, by reduced illumination, blurring of the vision, deviation of the rays to the periphery of the retina, parallax test, cover test, etc., if we would know the full and true muscular power of the eyes in the field of fusion; nevertheless, all of these various procedures are useful as confirmatory tests for any muscular imbalance shown by what I consider much more practical and standard tests. We must consider all the various conditions that control the movements of the eyeballs, apart from their special function. The nervous supply controlling the movements of the eye consists of no less than four of the cranial nerves, one-third of the entire number, with that of the special function, convergence, controlled by a special center, lying in the mid-line of the brain and in the region of the Aqueduct of Sylvius and the fourth ventricle. This special nucleus while it seems to control the action of convergence, does not produce nor cause exophoria when paralyzed. Furthermore, may we not consider the actions of the eyeballs as those of control, directed by the higher centers of the brain, the hemispheres, as shown in the field of version and those which are automatic, directed by the lower brain centers, as the Medulla and the Pyramidal Tracts. These lower centers seem to control the act of fusion and that of convergence. Hence I cannot favor any test that will disturb these important functions. Now there is a standard of all these functions of the eye. It is invariably found in the normal eye and must exist in the vast majority of all human beings. It is the typical eye of nature and under all reasonable conditions this eye will never suffer from muscular asthenopia. Before proceeding with the method of using the prism test I wish to state that the power of abduction as shown by this test is not always the key to a solution of our problem nor is it a fixed condition,

as under certain procedures I have found the power of abduction to vary very much from the first tests. In the use of the prism test with the base in, testing the power of the externi to rotate the visual line outward under the guiding sensation, the patient should be able to fuse or blend the images of a candle, placed at twenty feet with a prism of eight degrees or centrads. This is about the normal power of the externus, though it may vary from this and yet be in perfect balance according to the power of adduction. If the eight degree prism can be overcome by the eye turning outward then we increase the angle of the prism and if it rises as high as ten, twelve or more degrees we have probably an exophoria. Now repeatedly test the power of adduction (the normal power of adduction is about twenty-four degrees at the first or second examination) and if, after these repeated trials we can only get fusion of the images with a prism of less deviating

MADDOX COMPOUND ROD.

MADDOX SIMPLE ROD.

power than that of the externi the evidence of exophoria is almost complete, particularly if the field of fixation or version shows the same outward tendency of the visual lines. I place almost complete reliance on the findings of these two tests, at the same time I use as confimatory tests first the power of convergence as shown by the fusion near-point, the cover test, to note the rotation of the eye when deprived of the visual power, and the Maddox rod test or the old vertical diplopia test to note the devi-

ation of the eyes when deprived of the fusion power. These last tests are very useful in prescribing prisms.

Treatment. In my experience the correction of exophoria is one of the most difficult problems in ophthalmic procedures,—the fusion force of the eye is so low in the field towards the nose ; the power of the externi to turn the eye outward in the field of fusion is very small ; the muscle itself when operated upon is generally found more or less relaxed, nor does it respond to orthoptic training as readily as the interni, if at all, and the use of glasses seems to have very little effect on the tendency of the eyes to turn outward. But in all cases of evident exophoria as shown by the tests for that condition, if they have not been supplied with the correct and suitable glasses, I would first fully correct any refractive error that may exist, under a mydriatic, if necessary. Let these glasses be worn constantly for some time, though I do not believe that any refractive error is the cause of the exophoria, yet the full correction of a myopia, from the more distinct retinal images formed, must increase the fusion power of the eye and in this way we may have an increase in the power of adduction. I think this will only apply to cases of myopia, while, on the other hand, in exophoria associated with other refractive conditions glasses will be of very slight service.

The use of tonics, as Strychnia, etc., and out-of-door exercise may be indicated in some cases, particularly when following any severe illness, yet even these cases may show fairly good adduction and convergence and the improvement will only be in the general condition. Our next step in the line of treatment may be that of orthoptic training, by prism exercise, for the interni, two or three times a week. As to the method with which this exercise shall be used there seem to be as many suggestions as there are writers on the subject. In my

experience the stimulation of the interni by the use of a weak prism placed over first the one eye then the other, with the base outward every few seconds as the images unite, for five minutes, has been serviceable, but, as Winters well says, "Muscular power developed in this way is not permanent," as when the exercise is stopped the original condition soon returns and the symptoms reappear. Before proceeding to the consideration of an operation there yet remains the method of prescribing prisms for constant use placed with the base of the prism over the internus. I do not fully approve of this procedure, yet in some cases, where we meet with a decided objection to any operative interference, they may be tried. For this purpose, after the degree of the outward tendency has been carefully estimated with Maddox's rod or the vertical diplopia test, we may order prisms of equal degree for each eye. There seems to be some limit to this, as few patients will tolerate more than a correction of six degrees of exophoria with a prism of two or three degrees over each eye. Still the use of prisms is only a temporary condition; as the effect is not permanent, they tend to develop and to increase the tendency to deviation and simply postpone the time when we must come to the final and permanent result of an operation. All these methods should be given a reasonable and serious trial before we approach the proposition of an operation. Failing then in all our procedures to correct the symptoms or to produce a constant and fixed result, we not only must decide on an operation, but also the very important question, What operation to perform? Here, in my experience, comes a very serious question, as we must decide whether our case of exophoria is due to a weakness of the interni, deficient adduction, or increase in the power of the externi, increased abduction. If we have the latter condition, that is to say if we find the abduction

very high, of ten or more degrees of prism deviation, then we may consider the power of the externi as too great and a tenotomy, partial or complete, on one or both externi, will be the first steps in our operative procedure, to be followed, if necessary, by a strengthening of the interni. Conversely, if we find the abduction eight degrees or less, with the same tendency to exophoria, then any procedure that will tend to weaken the power of the externi is not indicated, but we must increase the power of the interni by a suitable operation. For this purpose I think the operation for shortening of the interni, one or both, is the best and safest procedure. This operation of a "tuck" in the interni was originally performed by me with the catgut suture and has seemed to me to be the best, most rational and most certain in its effects on the muscular balance. This operation is well known, having been many times described by others, with the use of the silk suture, which requires another operation to remove it, and by myself with the use of the catgut suture where the advantages are obvious. This procedure is simple in its technique, easy of performance and very gratifying in its results, while being perfectly free from any danger of overcorrection. As to the advisability of an advancement of the tendon of the interni in these cases it has seemed to me too much of a formidable operation and is not needed nor indicated in these conditions of imbalance. Stevens says: "Every modification of the length of a lateral muscle, by relaxation or by shortening, which can in any degree effect the rotation of an eye, must in every instance be accompanied by an exactly equivalent modification of the corresponding muscle of the other eye." This rule does not meet my full approval as while it may and probably does hold good is most cases of exophoria that require an operation, as the increased effect is generally needed, yet it seems to

me and has been so in my past experience, that the extent of all our operative procedures must depend on the extent, or degree of the tendency to a deviation. That is to say, if the evidence of an exophoria is very slight and yet needs an operation for its correction, we may very reasonably reduce the power of an externus or increase the power of an internus and then note the effect on the field of fusion and on that of fixation. If this simple procedure has been sufficient surely no further operative interference will be needed. I have the records of many cases where only one operation has been performed and an examination after several years has shown that the result had been permanent. The effect of an operation on one lateral moving muscle is almost invariably shown in the power of the corresponding muscle of the other eye or has been so in my experience as shown by many records and I have considered this due to the physiological action of binocular adjustment of the visual lines. This is well demonstrated in the permanent correction of convergent squint by a simple tenotomy of one internus.

Conclusions: That exophoria is frequently met with in ophthalmic practice in the investigation of the refractive condition, as shown by the examination with the tropometer, the prisms and the cover-test.

That the primal cause of exophoria is an antecedent weakness of the interni and, in some cases, an increase in the power of the externi, both conditions being probably congenital.

That it is very difficult to bring about a perfect adjustment of this imbalance of the ocular muscles, but that a careful adjustment of this imbalance will give constant and decided relief, and in the majority of cases an operation is to be preferred to all other procedures as full and perfect relief is not always obtained by the orthoptic training of the interni, prisms, or the constant use of glasses.

CONDENSED HISTORIES OF CASES OF EXOPHORIA.

No.	Vision Rt.	Vision Lt.	Age	Refrac.	Add. first	Abd. ex.	Tropometer in	Tropometer out	Operation tuck int.	Operation ten ext.	Exoph. deg. before op.	Add. last	Abd. ex.	Glasses	Remark years after
1	15	15	40	Hy.wAh.	8°	8°				yes		15°	4		
2	20	40	24	Am.	5°	10°				both				yes	
3	15	15	49	Ah.	10°	10°			L.			12°	8°		
4	15	15	25	Hy.	4°	4°			R.		3°	12°	5°		
5	15	15	8	Hy.	6°	8°			L.			15°	8°	"	
6	15	15	23	Ah.	10°	10°				R.		15°	10°		
7	15	15	53	Hy.	8°	12°				L.	3°	15°	10°		2
8			17	My.			50–50		R.						
9	15	15	27	Ah.	6°	10°	45–50 to 45		both		10°	20°	8°	"	
10	70	15	19	Ah.	2°	10°	35–50		R.			15°	3°		
11	20	20	43	My.	12°	12°	40–45		L.					"	
12	15	15	16	Hy.	8°	12°	45–50		Rt.			15°	4°	"	
13	20	20	42	Hy.wAh.	10°	10°				both	3°	12°	5°	"	
14	15	15	35	Hy.	5°	5°	40–40		Lt.		3°	15°	4°	"	
15	30	40	44	My.wAm.	15°	10°	40–45			both	3°	25°	10°	"	
16	20	50	15	My.wAm.	12°	15°	50–50		Lt.			15°	6°	"	2
17	25	40	32	Hy.	10°	10°				R. Sup. & R.		15°	8°	"	3
18	15	20	41	Hy.wAh.	2°	15°				both		10°	8°	"	
19	15	15	26	Ah.	8°	12°				Lt.	4°	20°	8°	"	2
20	20	50	48	Hy.wAh.	10°	10°	R. Hyper.			R. Sup. & L.	6°	15°	6°	"	2
21	15	15	25	Hy.wAh.	6°	8°				both		15°	5°	"	
22	15	15	27	My.	10°	15°			Rt.	both	15°	20°	10°	"	
23	15	15	33	Ah.	10°	8°			Lt.	both		15°	5°	"	
24	20	20	20	Hy.	15°	8°				Rt.		20°	5°	"	
25	15	15	28	Hy.	10°	8°				both		20°	6°	"	
26	15	15	15	Ah.	8°	8°				Lt.		15°	8°	"	
27	15	15	30	Ah.	8°	8°				Rt.		20°	6°	"	
28	40	20	40	Hy.wAh.	10°	12°	40–50			both		20°	5°	"	
29	15	15	13	Ah.	12°	15°			oper	ation ?	6°	25°	10°	"	
30	30	30	28	My.wAm.	8°	10°				Rt.	5°	15°	6°	"	
31	15	15	30	Hy.	10°	10°				Rt.	8°	20°	5°	"	1
32	20	20	22	Am.	8°	15°			Rt.		5°	30°	8°	"	
33	40	15	38	Am.wAh.	6°	15°	45–50			both	10°	20°	10°	"	
34	15	15	43	Hy.	8°	10°				both		12°	8°	"	
35	100	20	27	My.wAm.			R. Hyper.		Rt.	Rt.	5°			"	5
36	15	15	24	Em.	8°	8°			Rt.	Rt.	12°	25°	8°	"	
37	30	30	26	My.	5°	15°			Lt.	both		20°	8°	"	
38	15	15	35	Hy.wAh.	8°	8°				Rt.		10°	5°	"	4
39	15	15	27	Ah.	6°	8°				both	5°	12°	6°	"	
40	15	15	31	Em.	10°	10°				Rt.		20°	10°	"	
41	20	70	35	Ah.	2°	2°	R. Hyper.		Rt.		6°			"	
42	15	20	32	Ah.wAm.	12°	16°				both	15°	10°	10°	"	
43	15	15	26	Am.	12°	10°				Rt.	10°	15°	8°	"	4
44	15	15	26	Em.	10°	10°				both	1°	20°	6°	"	
45	15	15	25	Ah.	4°	4°			Rt.			10°	4°	"	2
46	20	20	27	Hy.	10°	10°				Lt.	1°	20°	6°	"	
47	15	15	13	Hy.	4°	4°			Lt.			8°	4°	"	1
48	20	20	37	Ah.	12°	8°				Lt.		12°	8°	"	
49	15	20	55	Hy.wAh.	4°	10°				both		10°	6°	"	
50	15	15	26	Ah.	6°	8°	45–45		Lt.			15°	5°	"	3
51	15	15	38	Hy.wAh.			R. Hyper.			R. Sup. & L.	15°	orth.		"	12
52	15	15	24	Hy.wAh.	8°	8°				Lt.	3°	15°	5°	"	
53	15	15	32	Ah.	8°	8°				Lt.	6°	15°	6°	"	
54	15	15	38	Ah.	12°	10°				Lt.	4°	15°	10°	"	7
55	20	20	30	Am.	2°	10°			both	both	8°	15°	15°	"	4
56	15	15	38	Hy.	6°	8°				both	10°	15°	8°	"	4
57	15	15	23	Hy.wAh.	10°	10°				both		15°	5°	"	
58	15	15	25	Hy.wAh.	10°	10°				both		20°	6°	"	4
59	15	15	30	My.wAm.	10°	10°				both		12°	5°	"	
60	20	20	37	Hy.	12°	10°			Rt.			15°	8	"	

These cases taken from my office work have all submitted to one or more operations as needed. They show some interesting conditions and conclusions in reference to the prism test for the condition of exophoria. I particularly wish to show the results of the examinations in the field of fusion as I consider the prism test in this field the most reliable as to an imbalance of the ocular muscles and at the same time the best test for the indications for our operations, also for the natural balance of the muscles of the eye. The operation of tenotomy of the externi was performed much oftener than the operation for shortening of the interni with the catgut suture, but only because some of these cases were operated upon before I commenced to do the shortening operation, and while the final results were very gratifying, in many of these cases, at the present time, I should prefer the shortening of the interni to that of weakening the opposing muscle. An interesting fact in connection with these tables is that the greater portion of them do not show myopic refraction as nearly three-fourths of them are hyperopic with or without astigmatism. The figures for the field of fusion are the final ones, that is to say, the results of the examinations of adduction and abduction after several trials with the prism test and in which there was no increase in the power of adduction by the exercise of the interni. They compare very favorably with Bannister's tables. The visual power is noted by the lower figures of the usual notation and is readily understood by adding the numerator 20 to those given in the tables, as 70 indicates V. = 20/70, and 15 indicates

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Explanation: Visual acuity, the numbers are the denominator with 20 as the numerator in all cases. Prisms were used in some cases but failed. Results: Two, failures. Nineteen, improved. Thirty-nine, decided improvement. Twenty-two, shortening by catgut suture. Twenty, single tenotomy. Twenty-one, both externi tenotomized. Three, tenotomy superior. All of them wore glasses for a reasonable time and the visual acuity is with glasses. Four showed hyperphoria.

20/15., etc. In the other columns will be found the refraction, age, operations, tropometer findings, time elapsed between the first and final examinations, and lastly the test for adduction and abduction by prisms. All these cases show exophoria more or less before operation and orthophoria after the final examination.

Paralysis of Convergence: Although not connected with the subject of this work, yet, as this condition may be mistaken for a slight divergent squint or exophoria, I deem it necessary to mention its essential features. In the discussion of exophoria I have spoken of the center for convergence and of its location and of its action on the eyeballs, in which we find that it controls convergence of the visual lines from the first position to that of the most extreme convergence that the action of the interni will admit. That the control of this center is limited to this action is well shown in the cases of paralysis of this function, as we find very slight, if any, crossed diplopia — or it may be homonymous — there is no appearance of any deviation of the optic axes, though in some cases a slight deviation may be detected under an examination with the perimeter. The slight inability to fix the eyes inside of the point of infinity may give rise to some unsteadiness in walking. In the field of version we find the associated movements of the eyes are *almost perfect*. They will move in unison to all parts of the field, but as soon as the test object is brought nearer to the eyes, diplopia commences and gradually increases. This shows crossed diplopia, becoming greater as the object is brought closer, and if the eyes are watched they will seem to stand at the first position, showing no power to converge. The field of fusion seems to be abolished, as adduction and abduction will be very low in degree, possibly only one degree in each direction. All this will cause a complete inability to read or to see near objects, as they

cannot fix with either eye an object held in the median plane of the head. In squint, one eye will always fix the object, no matter where it is held and the vision will be steady. An attack of this condition generally comes on suddenly, and must be due to some pathological condition in the region of the Aqueduct of Sylvius. It is then evident that the cause is central, and the only treatment that may be of service must be in that direction, while operative interference will not be of any use.

Paralysis of Divergence. This term is only mentioned in this work because it is frequently spoken of by other writers and for the sake of completeness. It seems to be used in the same sense as that of insufficiency of divergence, but the last named term is simply and only esophoria, or weakness of the externi. In my conception of this condition at the present time I must state that the condition of paralysis of divergence cannot occur, in either a natural or a pathological condition of the eyes, as I understand the action of the ocular muscles. I have only seen one case reported, that by Duane, in the proceedings of the Ophthalmological Section of the Academy, published in the *Archives*, May 1903, but I can see no evidence of a paralysis of divergence in this case.

Divergence of the eyes — as I understand it, and as I have discussed it in the subject of the movements of the eyes — consists of the motion outward of the visual lines, not from the position of extreme convergence, as some others claim, but from the first position of the eyes, when the look is directed forward to infinity, on or slightly below the horizontal plane; or, we may say, in other words, the position of the optic axes in complete paralysis of all the straight muscles of the eye, ophthalmoplegia externa.

Then divergence — being the ability of the eyes to move or rotate outward from this first position would place the

eyes in an abnormal position or in a position that is not required by nature and cannot exist as a special function of the eyes. It can only be present when there is some anomaly of the ocular muscles or when the condition is brought about by some artificial means, as by the action of prisms with the bases placed inward. The turning outward of the eyes, from the position of convergence, is not divergence, but simply one of relaxation of the interni from the stimulation of the convergence center, with the eyes returning to the natural or first position, and no requirements of nature or of physics will ever demand that the eyes should pass beyond that position, nor has any evidence ever been presented, as far as my information extends, that would show a possible center for that control or necessity for any function of that nature. The eyes may turn out, either singly or combined, under the action of prisms and the stimulation for fusion, but this is shown to be the direct action of the externi, as demonstrated by the tests for abduction. Hence, because we find an eye that from weakness of the externi cannot turn the visual line outward more than one degree of prism — the same as we meet in the cases of esophoria — this is not paralysis or even paresis and the truth of this proposition is easily demonstrated by the examination of the rotation of the eye in the field of version.*

I am inclined to think it would be better to eliminate the term of "paralysis of divergence" from those of the motility of the eyes, at least until some more positive evidence has been shown that we may have a center of the brain that controls this function. This so-called paralysis or esophoria admits of operative interference and cure. Its opposite or true paralysis of convergence will not be improved by an operation.

* See article by Berry, in *Trans. Oph. Soc. of the United Kingdom*, Vol. XXI.

CHAPTER VI.

HYPERPHORIA AND HYPOPHORIA, OR LATENT VERTICAL SQUINT.

These two terms signify practically the same condition of the visual lines, that is to say, a condition in which the visual line of one eye tends in a direction above the visual line of the other eye. This vertical tendency may exist in only one eye or the same tendency may exist to the same degree in each eye. This condition of double hyperphoria is called anaphoria when both lines tend to turn above, and kataphoria when both lines tend to turn below the plane of regard. It is in the study of these tendencies that one is led almost positively to the consideration of the muscular origin of all squint, and it is very difficult, I might say impossible, to find a dividing line between heterophoria and squint. If this be so then we must consider that all squint is simply a manifestation of what was a latent condition. This is well illustrated in hyperphoria, in which cases are reported of one degree up to ten degree or more and when beyond that then the tendency becomes obvious, although they may have binocular vision.

Hyperphoria and hypophoria, therefore, as the terms imply, denote respectively a tendency of one visual line to turn above or below that of the other, and may be designated as right or left according to the eye which tends to deviate from the plane of regard. Right hyperphoria denotes a tendency of the visual line of the right eye to turn in an upward direction, so that direct rays of light

will fall upon the upper part of the retinal field and be projected downward. The same rule will apply to all other deviations as proposed in the classification of this condition. These tendencies are not incompatible with perfect binocular vision as long as the fusion power is fairly well developed and with good illumination of the test object; but as soon as we reduce the fusion power by a colored glass, or the rod test, then the latent tendency becomes manifest. Diplopia will result according to the anatomical condition of the muscular structure of the eye Hyperphoria is very frequently found in the first examination of asthenopic cases, particularly if the diagnosis is made from the finding of the rod test or that of the phorometer, and is said to be about thirty or thirty-five per cent. of all the cases. This is a statement that I have no wish to deny, but as I have stated before, the rod and phorometer tests deprive the eyes of their most important and controlling function; consequently, I doubt very much if we can rely upon a diagnosis made in this manner. From this the inference is very obvious, that unless the deviation is very pronounced — five or more degrees — we may disregard any slight tendency of a vertical deviation when the fusion force of the eyes is abolished.

The etiology of all these conditions of vertical imbalance of the eyes rests simply on the power of certain muscles to turn the eyeball in a vertical direction, and not on the amount of innervation. If we seek for the primal or fundamental cause I think we will find it in an essential weakness of certain muscular structures that should tend to keep the eyes in the primary position when the fusion force of the eyes is active. "It is a local physical deformity," truly idiopathic and manifested by a weakness of some one or more of the straight muscles of the eye. Innervation, hypermetropia, or any other of the contributing causes of squint cannot play any part in these devi-

ations ; hence a vertical tendency, either up or down, is due to an essential weakness of one of the muscles.

To particularize this condition, we have in hyperphoria a weakness of one of the depressors of the eyeball, an inferior rectus ; in hypophoria, a weakness of one of the elevators of the eyeball, a superior rectus ; in anaphoria, a weakness of both inferior recti ; and similarly, in kataphoria, a weakness of both elevators. It is possible that we may find the reason for these deviations in an overaction of the muscles, but I prefer to consider the fundamental cause to be one of weakness, or deficient power in the anatomical construction or insertion of the muscles, for the reason that has been explained.

The symptoms of hyperphoria or hypophoria are very similar to those of the other forms of heterophoria — pain in the head being the most prominent ; sometimes extending backward to the occiput, with dizziness and nausea, and frequently felt in the mornings on waking from sleep. Irritation, with congestion of the conjunctiva, is frequently present, and diploplia may be occasionally noted. The habit of tipping the head is sometimes noticed in hyperphoria, so much so in some cases as to resemble wryneck or torticollis. In cases of anaphoria or its opposite, kataphoria, we have a tipping of the head either backwards or forwards. In anaphoria with a weakness of the inferior recti, both visual lines tend to deviate upward, the head is thrown forward to relieve the strain on the weak muscles, with contraction of the anterior portion of the occipitofrontalis, causing a constant scowl on the face. These cases are very miserable until the imbalance is corrected.

Diagnosis. From what I have stated in reference to the reliability of the different examinations for heterophoria and squint, there are only two tests that can be fully depended upon for the diagnosis of these vertical tendencies, all others being complementary. No operation should be

performed until these two tests fully agree upon repeated examinations! In vertical squint — hypertropia — the condition is obvious, but though we may note that the direction of the opposite axis is above that of the fellow eye, yet we must decide as to the rotational power of the muscles before we can have positive indications for our operative interference.

To illustrate the method of diagnosis in all vertical tendencies I will give the examination of the muscular condition of certain cases that will make the indication clear and the diagnosis to be readily understood.

Miss R. C., age 15. Refraction Hy. with Ah. 90°, said to have had convergent squint when a child, but at present the visual lines seem normal. Adduction R. 10°, L. 12°. Abduction R. and L. 5°. This duction test shows weak externi as the power of adduction may increase by testing. Supraduction, R. 1°, L. 3°. This vertical test for duction shows a decided tendency for the left visual line to tend upwards, at the same time the fusion force is fairly good. Tropometer, R. 30° up; 60 down; 50° in; 60° out. L. 40° up; 45° down; 60° in; 45° out.

In this examination with the tropometer we have a clear exposition of the imbalance of the ocular muscles in the left eye almost identical with that of the prism test, as this case shows the vertical tendency upward of the left eye from weakness of the inferior, and a tendency to esophoria in the same eye. The inference is plain in this case that the child probably did have convergent squint of the left eye, but as the fusion force became more fully developed with age the visual lines have become adjusted, binocular vision is re-established, and there is no evidence of squint. This case well illustrates the power of the fusion force as a contributing cause of squint, with a tendency of the eyes to look to the right, not shown in true lateral squint, and the reason for the old story "the child will outgrow its squint."

Another similar case, but more pronounced, that cannot outgrow the squint, is the following:

Master R. A. F., age 14, has a fixed upward squint of the left eye, deviating about two millimeters. V.=20/15 each, no glasses accepted, possibly +. 50. cyl. ax. 90°. No binocular vision, and consequently no fusion test with prisms.

Tropometer = R. 30° up; 55° down; 50° in; 50° out.
" = L. 45° up; 40° down; 60° in; 45° out.

Here again we find a clear, positive indication ot a muscular imbalance, similar to the one first mentioned, but we also notice the extreme weakness of the left inferior; hence though the boy has a similar imbalance, his squint remains permanent. In this case I shortened the inferior — no indication for tenotomy here — with the following result:

Tropometer. R. 35° up; 50° down. L. 30 up; 40° down. Lateral balance same as at first. Subduction 4° each. This boy has binocular vision at 20 feet and with the stereoscope.

Third case. Mr. T. H., age 39. This man has suffered for a long time with a constant tendency to scowl and to hold the head forward, as he can see better in this position. He has constant pain in the head and photophobia. V. =20/50 w. + 50.= 20/20 each. Adduction 10°; abduction 4°; supraduction 1° each. His vertical rotation measured by the perimeter shows right eye up 33°; down 20°; left eye up 40°; down 20°. By the

Tropometer =R. 30° up; 45° down; 50° in; 50° out.
" =L. 30° up; 40° down; 50° in; 50° out.

His lateral movements of the eyes were normal. In this case, as the superior seemed to be so powerful, I did a complete tenotomy of both elevators, with complete and permanent relief of all symptoms, and the objective examination was as follows,— tropometer, each eye, up 25° and down 50°; lateral balance the same as before. Here I considered that a tenotomy of both superior recti would correct the condition better than a shortening of the inferior.

In the consideration of hyperphoria, an important question arises as to the influence that this condition may have on the lateral balance of the ocular muscles. Stevens at one time claimed that all lateral imbalance and even fixed squint was due more to a vertical tendency of the ocular muscle than to any lateral deviation that may be present. While I cannot agree with this suggestion — as in many cases I have seen a vertical tendency disappear under the correction of a lateral imbalance by an operation — yet the vertical balance of the eyes is very important, and should be carefully considered in all our cases. This is well shown in the test with prisms, as in the estimation of adduction and abduction it is very essential that the prisms should be held or placed in the frame perfectly horizontal, particularly so when using the higher degrees. The slightest tipping of the apex of the prism will so deviate the image of the test object beyond the slight power of the vertical acting muscles, that the images will not fuse, but they will readily do so when the images are perfectly level. It is, therefore, necessary to see that the images are on the same plane if we would find out the true power of rotation of an eye in the horizontal plane.

The power to turn the eyes up or down under the fusion force is very low, only from one to three degrees, and this function may be very easily disturbed in the presence of a lateral tendency. Many cases will show a much larger power to rotate in the horizontal plane by careful attention to this point. On the other hand, the necessity of holding the prism perfectly vertical in testing superduction and subduction is not so necessary owing to the greater power of the lateral moving muscles.

In many cases we may have a vertical tendency associated with a lateral deviation, and the question arises as to which condition should be first corrected. The answer to

this will depend upon the degree of deviation or tendency in each direction. In other words, if we have a decided vertical tendency to deviation, then this condition should be corrected first. If I find by the rod test a vertical tendency of three or more degrees, I would give this very careful attention, and in this consideration we have the very important question — where is the essential fault? Is it in the superior or the inferior that the weakness may be found? The test with the rod will not show it, nor that of the phorometer, but it can be well demonstrated by the careful test with prisms. This must be and can be scientifically confirmed by the tropometer. The measurements by this instrument are very exact and at once indicate the muscle that is essentially too strong, or the one that is essentially too weak. A decided rotation upward of less than 30° will show weakness of the upward turning; and downward, if less than 50° will show a weakness of the down turning.

Furthermore, if we do find a deficiency of power in the vertical acting muscles, then the consideration is presented which muscle shall be corrected? In other words, is it more important to weaken a strong superior recti or to strengthen a weak inferior recti? It goes without saying that one of the most important functions in the field of version is that of looking downward, as in all our work the visual lines must be carried and held below the horizontal plane. For this reason, unless the upward tendency is very great, I prefer to make the muscle of subduction stronger by the operation of shortening the inferior to that of weakening the opposing superior rectus. A case that will show a vertical tendency, in which we find the lateral fusion force in the proportions of one of abduction to two or three of adduction, will always be improved by a correction of the hyperphoria, but in my experience, if the lateral muscles show an imbalance of

one of abduction to one of adduction (exophoria), or one of abduction to four or more of adduction (esophoria), then we may correct these conditions, even in the presence of a slight vertical tendency, with almost positive assurance of success.

There are still other cases that may come under this classification, not infrequent, in which we have a tendency of both visual lines to turn the same way but laterally, in other words, they tend to look to the *right or left.* I have no terms to express this condition nor do I think any have been suggested, but these cases are occasionally met with in an examination, and Stevens has suggested that it is due to a condition of cyclophoria. I have not convinced myself that that suggestion is correct but I am inclined to look upon all these cases as due to the same conditions as in all other cases of squint, that is to say, we have an essential weakness of one internus associated with a similar weakness of one externus, hence the tendency to look in one of the two lateral directions.

The diagnosis of this condition can only be made in the field of version, as shown by the tropometer or possibly by the examination with the perimeter. I will illustrate this by two cases.

Mrs. A. J. S., age 42. V. = 20/15. Hm. +1. D. Glasses assist her very much but still she has some pain in the back of the head and neck. Add. 12°; Abd. 10°. Rod, esophoria.

Tropometer =R. 30° up; 60° down; 40° in; 50° out.
" =L. 30° up; 60° down; 50° in; 40° out.

It is evident that the right eye shows a tendency to turn outward and the left eye to turn inward; in other words, a tendency to look to the right. We also note that the prism test shows exophoria and the rod test esophoria.

Another case:

Miss H. D., age 24. V.=20/15. Ah. 90°. Her sister states that this young lady is always looking to the right.

Tropometer=R. 30° up ; 50° down ; 50° in ; 55° out.
" =L. 30° up ; 56° down ; 62° in ; 50° out.

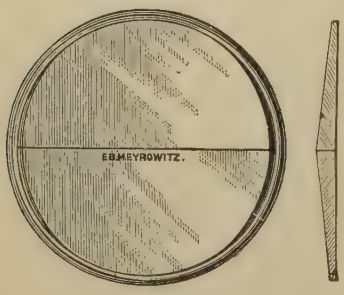
It seems to me that the treatment of these cases is the same as in all other cases of imbalance, according to the indications, as by a tenotomy, partial or complete, or by a shortening if needed after the correction of the refraction. In these cases the prism test of the fusion force will not indicate the imbalance as the slightest turning of the head will place the eyes in such a position that the relation of adduction and abduction may be almost normal. The rod test may give us some indication of an imbalance, as in one of the above cases we had an esophoria of 7° that was completely corrected by a shortening of one of the externi.

Cyclophoria. It does not seem to me that this work will be complete without a description of this anomalous condition of the ocular muscles ; although it does not produce a squint, it may complicate an imbalance. It is very seldom met in the examinations, and if present is probably due to a paresis of one of the obliques. Savage and also Stevens seem to place very great reliance on the influence of torsion in the estimation of an imbalance of the ocular muscles, but as far as my own examinations show I have failed to find it, unless associated with an astigmatism with oblique axes or with some other condition of heterophoria, the correction of which has seemed to correct the fault in the torsion of the eyes. As the term is expressed it represents a tendency of the eyeball to turn on the optic axis, the upper part of the vertical meridian tending to turn, either outward or inward, in reference to the median plane of the head or to each other.

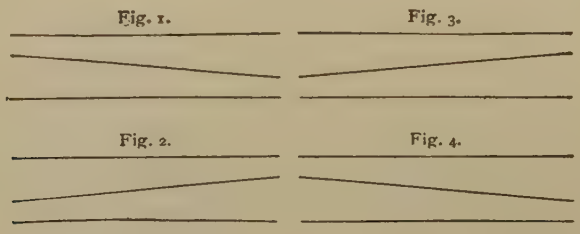
The necessity to keep the vertical plane of one eye parallel to that of the other or to the median plane of the head in all the movements of the eyes in the binocular fields of vision is too obvious to need any explanation. Its

importance in the act of binocular vision is very great, yet it seems to me that the necessary power to rotate the eye about the optic axis is very slight and consequently the liability of any disturbance from this cause can be almost neglected. The terms suggested to denote this condition are: Plus Cyclophoria, by Price; Plus Declination, by Stevens; and Plus Torsion by Maddox, when the vertical axes of the eyes tend to turn away from the vertical plane of the head. When the vertical axes tend to turn toward the median plane we have Minus Cyclophoria, etc. It is to be regretted that these writers did not select the same terms, but I think that of Price is the best as expressing the conditions.

Diagnosis. For this purpose a Maddox double prism with the bases horizontal and the test object a line on a white cardboard is used, when testing this function at the reading distance or about twelve inches. This prism, when placed over the one eye, will cause the line to be seen double and the line that is seen with the other eye will be found between the others. If all three lines are seen parallel there is no tendency to cyclophoria, but if the central line tends to tip in any direction there may be some weakness of the obliques. The appearance of these lines in the various types of cyclophoria is shown in this diagram.



MADDOX DOUBLE PRISM.



Position of the lines in *a* under-action of the left sup. oblique
" " " " *b* " " left inf. oblique
" " " " *c* " " right sup. oblique
" " " " *d* " " right inf. oblique

The clinoscope, improved by Stevens, is an excellent instrument for the diagnosis of any weakness or paresis of the oblique muscles. It is shown in the illustration.

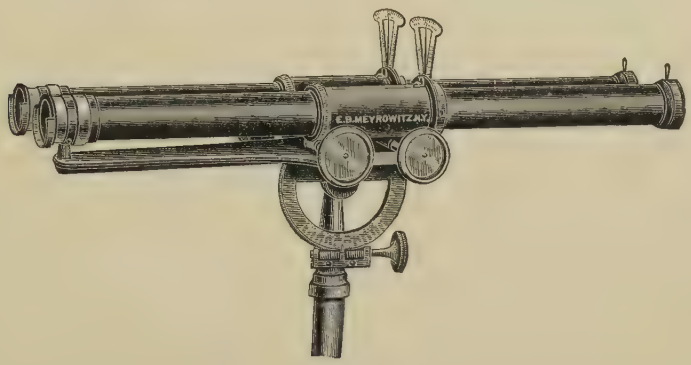


ILLUSTRATION OF CLINOSCOPE — STEVENS' LATEST MODEL.

The description for the use of this instrument is as follows: "The discs with a single pin, with the head in the center, drawn on each should be placed so that the point may be up for one eye and down for the other. Each pin should be vertical and the instrument so adjusted as to allow easy fusion of the heads of the pins. When the two discs appear as one the two pins should be the vertical diameter of the fused discs. If the one pin is a radius pointing in one direction obliquely, and the other is a radius pointing obliquely in the other direction (one toward the right and the other toward the left, making an oblique diameter) there is plus cyclophoria of one eye and minus cyclophoria of the other. If the upper pin is seen by the left eye and the lower pin is seen by the right eye, the two pins pointing to the right would show plus cyclophoria; while minus cyclophoria would be shown by the two pins pointing to the left. If the top pin is verti-

cal and the bottom one points to the right there is plus cyclophoria of the right eye alone, or if the bottom pin points to the left while the top one is vertical there is minus cyclophoria of the right eye alone, etc. When the pins are oblique the tubes to which the discs are fastened should be revolved until the two pins are vertical, forming apparently the vertical diameter of the fused discs. The index connected with each tube will point to the mark on each scale indicating the quantity of error in each eye." The treatment of these cases of cyclophoria may be by the rotation of cylindric glasses when there is oblique astigmatism, by the use of displaced cylinders, or by an operation on the tendons of the straight muscles. While the writers on this subject claim some very good results I have not had sufficient experience with their methods to advise its use.

All these varieties of imbalance, both vertical and horizontal, may be manifest or latent; that is to say, they may be manifest at the first examination or may be demonstrated by persistent trials of the action of the muscles. I have not found it necessary to make this distinction in my cases, as I think the present method of examination should fully develop any latent error in the balance of the ocular muscles that may be present. The constant testing of the interni may show an increased power of adduction, and the wearing of prisms, bases in or out, may develop an exophoria or an esophoria, but I do not think these procedures will change the rotation in the field of version. It is by the combination of all the true and confirmatory tests that we may arrive at an exact conclusion as to the imbalance of the ocular muscle with two or three examinations, and then we may direct our treatment or operation as indicated by these tests.

CHAPTER VII.

STRABISMUS OR HETEROTROPIA: CONCOMITANT OR FUNCTIONAL.

The extremely interesting paper on "The Etiology and Educative Treatment of Convergent Strabismus," by Priestly Smith, M. R. C. S., reviewed in the *Ophthalmic Record*, from the *British Medical Journal*, July 2, 1898, points to the innervation theory of squint for its etiology, as he states that "convergent strabismus is a disorder of innervation in which the visual centers fail to control the act of convergence," and also dwells on the old theory of relative hypermetropia with its influence on accommodation and convergence, thus placing all cases in the same uncertain etiology.

Donders' antithesis is as follows: "Hypermetropia causes accommodative asthenopia, to be actively overcome by strabismus convergens. Myopia, to muscular asthenopia, passively yielding to strabismus divergens." A very beautiful theory, which has found many admirers and disciples; but how many cases do we meet that completely refute and demolish these propositions?

Hansen Grut, in his prize Essay, advances the innervation theory for his cases of convergent squint, the position of rest for his divergent cases, and suggests that after death or in full ether narcosis, the eyes diverge; but in reference to this Schweigger says, he knows that neither "death nor narcosis is the natural condition of man."

My friend, Dr. A. A. Hubbell, has also advanced the innervation theory as his consideration of the "Patho-

genesis of Concomitant Strabismus," N. Y. State Medical Association, 1901, from which I quote: "At the outset I wish to define concomitant strabismus as a disturbance of oculo-motor innervation, accompanied by a perceptible deviation in any direction of the visual axis of one eye from the point of fixation of the visual line of the other eye."

Walfors says: "In my opinion squint depends upon an antecedent anomaly of the muscles, in which, under the influence of several accompanying factors, among which innervation plays an important part, carries the eye into a squinting position." I regret that I am not acquainted with Walfors' theory in full, but it seems to me that the first part of his proposition may lead us to a true theory.

Schweigger's articles do not seem to advance any particular theory, unless it is that of Donders, but refers to all in a general way, while Landolt also seems to hold to the antithesis of Donders.

Worth, on "Squint," P. Blakiston & Co., 1903, seems to place all his theory of the cause of squint on the loss of the fusion power, and claims certain results from the use of his amblyoscope, by which he has corrected a certain percentage of cases; but it seems to me that the per cent. of correction is about the same as that obtained with the use of glasses. He states (p. 168): "Heterophoria may be due to a muscle or group of muscles being too weak or too strong for the opponents or the abnormal insertion of a tendon . . . heterophoria is essentially a motor anomaly, but 'Squint,' on the other hand, is essentially due to a defect in the fusion faculty. In the presence of this fundamental cause, heterophoria may give rise to a permanent squint; not otherwise." Here, we have two causes for what is essentially the same condition, as numerous writers consider fixed squint a greater degree of heterophoria. Furthermore, Worth says, in reference to the muscle theory,

"It may seem reasonable to attribute this deformity to a defect in the muscles which move the eyes. A little investigation ought to convince any one of the falsity of this view." I quote this because twenty years' investigation has not convinced me; and I find that Worth bases his reasons why the muscles are not at fault on the ability of the eyes to follow the finger or pencil in the field of version and then to note the corneal edge in reference to the outer canthus, etc. He claims 81 per cent. perfect by this test but had he made the examination with the tropometer — a much more exact and scientific method — he would have found a deficiency in the field opposite to the deviation of the squint in every one of his cases or one hundred per cent. That is the result of all my examinations.

All these writers have advanced their ideas on the subject of the causes of squint, and one writer will directly oppose the theory of another, so that when one reads the diverse opinions of such eminent men in the profession of ophthalmology he feels somewhat at a loss to understand what are the true causes of squint.

Every physician must form his own opinion of the existing condition of the human body, either pathological or functional, from his individual experience in the results of his cases, and to formulate a theory must take many years of constant observation, together with a large number of illustrative cases. Hence, any theory must be so advanced that it may meet the indications in all cases, even though we may grant the proposition that "the exception proves the rule."

For several years I have watched, in all their varying conditions, my cases of strabismus, and I have felt that I could see and note a cause and effect based on the proposition which has been stated in a previous paper and further improved on by subsequent observations, as follows: That the eyes move under the innervation derived from

two centers of the brain — first, in the field of fixation, (controlled by the higher centers of the brain), in which all the straight muscles of the eye are capable of receiving nearly the same amount of innervation, and consequently, under the stimulation of the will-power turn the eyes equally, or nearly so, in all parts of the field of fixation; second, that the eyes rotate in the field of fusion, in various degrees, in which they do not move under the direct control of the will-power, but about a common point, in certain directions, under the stimulation of a center, controlled by the automatic action of the brain. This function is not influenced by the action of the will. This is called "unconscious innervation," and these centers receive their stimulation from the desire for fusion of the images on the retina. These movements seem to be controlled by the relative power of the straight muscles, as shown by their size and their insertion into the sclera.

Granting the above conditions as existing and controlling the movements of the eyes and the direction of the visual lines, we may construct a theory that will elucidate and explain the causes of all conditions of strabismus.

Hansell, of Philadelphia, says: "It is unscientific to attempt to separate the muscular from the innervational apparatus, the more so as the latter comprises both the fusion force and the impluse conveyed by the nerve trunks; and equally futile is it to speak of one muscle as having more power, as measured by prisms, than its corresponding muscle in the other eye." Having read this, I turn to Stevens and find: "All these determinations having been made with sufficient care (that is, the equilibrium test, etc.), the examiner proceeds to ascertain the relative power of the different pairs of muscles by finding the strongest prism with which images can be united in different directions." If, then, the prism test is unscientific and useless, why use it at all? In my opinion it is one of the

reliable tests for the relative power and the indications of a muscular anomaly.

May not a theory then, depend upon the muscular conditions of the eyes essentially, and which may be associated with certain other conditions in the eyes themselves; in other words, may not the position of the visual lines depend upon the fusion power, or guiding sensation of the eyes, and the *relative power* of the straight muscles?

If we study the theories and the propositions as advanced for the causation of squint, all cases must be placed under one head or class, dividing them simply according to degree, as latent, periodic, and fixed; and, though the majority of writers apply the same theory to all alike, to my mind, and because of my experience, there seem to be two distinct classes, with their respective conditions, that tend to cause the squint, but with one *underlying* cause in all cases. Into these two classes we may divide all our cases of squint, then proceed to develop a theory that will cover both classes in which the relative power of the straight muscles of the eye must and do play the most important part.

Then, as I have always claimed (and I note that other writers are now alluding to it) we must take into consideration the guiding sensation of the eyes, the essential fusion power with other contributing conditions, if we would find a reasonable cause for squint and its dependence on the relative power of the muscles.

Before proceeding further with the argument, let me, in a few words, give the conditions of two cases of squint found in girls of the same family.

Rebecca L., age 14, convergent concomitant squint, fixed in the left eye, onset when one year old. R. E. V. $=\frac{20}{20}$, L. E. V.$=\frac{20}{200}$, Ah.$=$1.D. ax. 90° in each eye. Ophthalmoscope and retinoscope show the same refractive condition. Operation, tenotomy on the left internus,

when seven years old, or seven years ago. To-day there is no evidence of the squint, the cosmetic effect is very good, the visual axes seem parallel, and she seems to fix with both eyes at the near point, yet the visual power is the same as at the first examination. Her sister Edith L., age 11, convergent concomitant squint, nearly always fixed in the right eye, onset when six years old. V.=$\frac{20}{20}$ in each eye, Hm. 1.50 D. in each eye. No astigmatism. Ophthalmoscope and retinoscope show about 5 D. of hyperopia. Has homonymous diplopia of 30°. Glasses were ordered for this case with complete disappearance of the squint.

I would also call attention to a case of divergent squint that I operated on a very short time ago, when on the exposure of the tendon of the internus it was found to be very small, not more than $\frac{1}{8}$ of an inch broad; while the externus of the same eye was found to be more than $\frac{1}{4}$ of an inch broad. This is pretty strong evidence of the muscular action on the cause of squint.

Now, it seems to me, when we compare the clinical history and the result of treatment in the two cases reported above there must be some cause acting in each case, to produce the squint, but in a different way. In other words, in each case a weakness of the externus with a contributory cause, — in one case the amblyopia, and in the other the relative hyperopia. Will the innervation theory or the antithesis of Donders include both of these cases?

Therefore I believe in the proper study of the causes of squint we must divide them into two great classes, as illustrated in the cases given above. Perhaps we may not be able to "draw the line" very perfectly between these two classes in all cases, but we can do so near enough for all practical purposes. As I have watched and studied my own cases I feel almost positive we can

do so, while I have verified my conclusions over and over again in my clinical work.

In May, 1894, I read a paper on Strabismus before the Medical Society of the county of New York, in which I made the proposition to separate the cases into two classes, and since then I have satisfied myself of the correctness of my proposition; but, at the same time, I would still further advance my ideas on the muscular theory. In both classes of cases I would also place the balance of power in the ocular muscles as the true and primal cause of all cases of strabismus, or as Walfors would have us believe, on "an antecedent anomaly of the muscles"; and that innervation, or the position of rest, has nothing to do with it. If I am not mistaken, this can be proved in all cases.

Now this balance of power in the action of the straight muscles of the eye, or, as Duane has so well classed it, "the power of *duction* as shown by the prism test," has been proved long ago by many observers. I would particularly refer to the tests made by my friend, Dr. J. M. Banister, Surgeon U. S. A., in his very careful examination of one hundred soldiers who never had any asthenopic symptoms whatever, and with vision $\frac{20}{20}$, or better.

This balance of power of these muscles in their relation to each other, as shown by the fusion power or guiding sensation, will even during sleep determine the position of the visual axes.

Placing then the muscular balance as the essential condition in the causation of squint, we also find other *minor* causes that will influence the position of the visual axes. First, congenital amblyopia in one eye; and second, relative hyperopia, as suggested by the first part of Donders' antithesis, or any refractive condition from that of emmetropia to the most complicated astigmatism.

I have now the memoranda of some 400 cases of latent

and fixed squint, all those in which the examinations were complete showing the same underlying cause. These cases I have divided as follows:

Esophoria	45 cases
Exophoria	68 "
Hyperphoria, right or left	8 "
Anaphoria	1 case
Cases looking to right or left	several

A total of about 130 cases.

Convergent squint, 1st class	93 cases
" " 2nd class	129 "
Divergent " 1st class	9 "
" " 2nd class	21 "

Cases in which the class could not be recorded, 23

This makes a total of over 400 cases that have been examined by myself and upon which my present opinions are based.

On these cases the following operations have been performed:

Advancements	4 cases
Shortening externi	72 "
Shortening interni	38 "
Total	114 cases.
Tenotomy of externus	105 cases
" " internus	94 "
" " inferior	4 "
" " superior	23 "
Total	226 cases

giving 340 operations on the entire series.

Now we will note the refraction of all these cases of squint, as far as the condition could be determined by the objective examination:

Convergent squint, Hyperopia	165 cases
" " Hy. with Ah.	34 "
" " Ah.	8 "
" " Myopia	6 "
" " My. with Hy.	1 "
" " Emmetropia	4 "
Refraction not recorded	25 "
In all	241 cases

Divergent squint, Hyperopia . . .	17 cases
" " Hy. with Ah. . .	5 "
" " Ah. . . .	8 "
" " Myopia . . .	7 "
" " My. with Am. . .	4 "
" " Emmetropia . .	2 "
Refraction not recorded . . .	6 "
In all	49 cases

The large number of cases showing hyperopia is due to the fact that many of my records are from the clinics at the Post-Graduate Hospital where the examinations were not very exact, otherwise I think that many of them would show hyperopic astigmatism. It is also remarkable to note the large number of cases of divergent squint that show hyperopia and astigmatism. As regards the sex of the cases, when recorded I found it to be about equally divided.

I will now call attention to what I consider determines these two classes, why they should be thus separated, and the causes therein acting to produce squint. In these classes I feel positive we may place all our cases of squint, whether divergent or convergent — (those cases with a vertical deviation need not be considered in classes) — and I hope to show some material advantage in so separating them.

In those of the first class we find this condition: Amblyopia, always congenital, vision less than $\frac{20}{100}$, associated with an abnormal balance of power in the lateral moving muscles. Hence with the loss of the fusion power or the guiding sensation the eye quickly turns in the direction of the most powerful acting muscle, namely, the internus, in convergent squint, and the externus, in divergent squint, etc. This is well illustrated by the early onset of the squint in cases of this class, and it is very clearly proved by the cases of amblyopia of the same kind that do not squint and which always show the same power of

abduction as adduction, or the same rotation in the field of version.

In the second class of cases we find this condition: Some refractive error, either hyperopia or myopia, with or without an astigmatism, and the vision nearly the same in each eye, but seldom less than $\frac{20}{70}$ in one and better in the other; and with this an "antecedent anomaly of the muscles" or, in other words, a weakness of some one of the straight muscles of the eye acting either laterally or vertically. *In convergent squint there is a deficiency in the power of the externi, and in divergent squint there is a deficiency in the power of the interni*, while the same rule will apply to all squints in the vertical direction. Cases of vertical squint are very difficult to prove by any other theory.

In the second class of cases, if the squint has existed for any length of time we may have what is called "*amblyopia ex anopsia*," arising simply from non-use and the suppression of the image in the squinting eye. I think these cases furnish the reports of the restoration of the vision after an operation, or after the accidental loss of vision in the fixing eye. (See article on Amblyopia). The theory as applied to this class is beautifully illustrated in those not infrequent cases of convergent squint with myopia, a condition that I do not think can be explained in any other way than that of a muscular anomaly.

The fact that all the various refractive conditions may exist without squint has been a very difficult problem to explain, or rather I have not been satisfied with the explanations that have been offered, so far as I have been able to consult the writers on this subject. Yet it is partly from this fact that I have come to my own conclusions, and I have yet to see and study the case that can not be clearly placed in one of these two classes and which will show an imbalance of the ocular muscles.

Fuchs, in the last edition of his text-book, gives the etiology of strabismus as follows: "Strabismus is, therefore, the result of the combined action of two factors, diminution in the visual power of one of the eyes and a pre-existing disturbance of the muscular equilibrium, according as the latter factor consists in a preponderance of the external or internal ocular muscles, a convergent or a divergent squint is produced," yet he considers *all* the cases of divergent squint as being myopic.

As it is utterly impossible to place them all under one general head, I would separate them into these two classes so that my theory will cover all cases, and in both classes we will find a certain weakness or want of power in one of the straight muscles of the eye.

Let me now resume the argument in reference to the first class, which I will illustrate with a typical case: A child, born of perfectly healthy parents, is normal in all respects — the visual axes are perfectly parallel, but the power of the external muscles of the eyes is not properly balanced. Now one eye is amblyopic, in which there is weak fusion power and partial absence of the guiding sensation in that eye, hence there is no tendency to fix the visual line (and I believe this power comes very early in life); then, with these conditions, it seems reasonable to claim that the eye will turn in the direction of the most powerful muscles. In convergent squint the eye turns inward and remains so, as there is no stimulation to make it resume its normal position. The same argument may apply to divergent squint under the same conditions.

Before proceeding further we must understand what this condition of amblyopia is: objectively we can find no evidence of a want of vision, for the examination with the ophthalmoscope gives us the same picture as is shown in the other eye, and as Maddox says, the macula appears

"tantalizingly perfect"; but subjectively there is all the evidence of the want of vision. These amblyopic eyes will not have a restoration of the vision under other conditions, no matter what may be the testimony to that effect by other writers. I make this statement because I have failed to ever meet a case; because a careful study of the various reports shows some flaws in the estimation of the visual power; and because other observers, just as careful and experienced, have made the same statement. Hence does not our clinical experience teach us that we *do* have a congenital amblyopia, not a suppression of the retinal images? For, just as soon as we can possibly test the vision of a child who squints, say at five years of age, we find it less than $\frac{20}{100}$; and waiting five years longer it is still the same. Can any one claim that there has been a loss of the physiological sensibility of the retina to reduce the vision from normal to less than $\frac{20}{100}$, in the first five years of life, and yet after that, or in the next five years, with the squint remaining, there has been no further loss of retinal sensibility and the vision remains the same? I would also refer to those cases where we have an amblyopia of the same kind, in which there never has been any tendency to squint and yet the vision remains the same through life. They seem to have binocular fixation, but careful examination will show *no* preponderance in power of any muscle. I therefore cannot understand the claim that this condition is amblyopia ex anopsia.

May we not forcibly ask: "Does all the loss of vision take place in the first five years of life and then remain so?" To me a negative answer is hardly required to such a self-evident fact.

Furthermore, if in the same cases the squint never is corrected it may remain until they are about thirty years of age and then the convergence disappears, the visual axes are parallel and remain so, yet we find the vision

about the same as stated above. This is the so-called natural cure of squint. It is very rare, and only confirms the muscular condition as the essential cause.

Moreover, examine those cases of monocular amblyopia who never had any deviation of the visual lines, and invariably you will find that the normal balance of power does not exist — that adduction is the same as abduction, that version of the eyes is normal, that there is no preponderance of power in either of the lateral muscles, and that the amblyopic eye simply follows its fellow in the associated movements. (See Amblyopia and the Restoration of Vision).

To repeat: What then are the conditions of our two classes of cases?

First, congenital amblyopia associated with an *abnormal balance* in adduction and abduction; taking from the eye its power of fusion or the guiding sensation, the eye must turn in the direction of the most powerful muscles — namely, inward or outward, up or down, as the case may be — the same conditions acting on the other eye under the screen test, for the visual lines maintain their respective distances, showing that the squint is concomitant or functional.

There must be a pre-existing disturbance of the muscular equilibrium in squint which can not be due to the eye assuming a position of rest, nor to the innervation, and, when the retina is deprived of its guiding sensation by the amblyopia, the eye must turn in the direction of the most powerful muscle. The above argument will apply to divergent and vertical squint, as in all these cases we have a muscular anomaly acting in the same way.

Second, in this class a child is born with perfectly normal vision in each eye. There is generally an error of refraction, either with or without astigmatism, and there is always an imbalance of the ocular muscles.

Now we notice, as the child plays with small toys or uses the eyes at a near point, that it begins to turn one eye inward at times — called *periodic squint.* This condition may continue until the child is five years old, when one eye constantly turns inward — called *fixed squint.* Does hyperopia cause this, when we see so many hyperopes that have no tendency to squint? May we not reasonably attribute it to an *insufficiency of the externi?* I think so, and this imbalance of the ocular muscles will explain the reasons for squint in cases of emmetropia, myopia, or astigmatism without hyperopia.

Now strabismus is generally convergent and the refraction hyperopic; why? First, because hyperopic refraction is the most common condition and second, because the centers for convergence and accommodation are very closely associated in the oculo-motor tract beneath the aqueduct of Sylvius and the floor of the fourth ventricle. Now as the child begins to use the eyes, if it is hyperopic it requires a greater power of accommodation. The correction of the hyperopia, by the increased accommodation, causes a stimulation of the power of convergence; then with an existing want of power in the *externi* we have the convergence of the visual lines.

Assuming this to be the condition, what becomes of the image formed on the retina of the squinting eye? First, the image of the object falls upon the least sensitive part of the retina — namely, the inner peripheral part — there forming an indistinct image; second, the image, whatever it may be, that is formed at the macula, or most sensitive part of the retina, is not in focus, the rays coming from a far different plane than the object, and again we have an indistinct image. Consequently the visual centers will readily suppress these indistinct images formed on the retina, and only single vision results. We can now say there is a loss of vision from "physiological

sensibility through psychical exclusion in the squinting eye," but we must remember that the sensitive retinal elements are still the same and may be developed to their former power when the eye is used under proper conditions. The vision is naturally reduced in one eye from disuse, the so-called amblyopia ex anopsia, and may be improved by an operation, by glasses, or by the exclusion of the other eye.

Strabismus has been divided into latent, alternating, and fixed, each and severally one of degree, and if so do they not all depend upon the relative power of the straight muscles of the eyes? It has been said that I "fall into error when speaking of the strength of the individual muscles estimated in this manner," referring to the test with prisms, and that "his argument seems illogical"; but if I am in "error," or "illogical," I shall be glad to be convinced of my fault. I have studied the cases of squint coming under my observation time and time again, both in my private and hospital practice, and have read all the literature that I can find on this subject, and I have only become more and more convinced that this theory of the action and the relative power of the ocular muscles is the chief and determining cause of all cases of strabismus. I have yet to meet with a case that cannot be explained in one of these two classes, and in which I cannot readily demonstrate the influence of the ocular muscles in the production of the squint.

Moreover, we meet so many cases with all the other essential conditions that we find in squint, and yet there is no deviation of the visual lines. Take, for instance, those cases of amblyopia congenitalis in one eye, perhaps hyperopic or astigmatic; no asthenopia; intelligent, bright people between fifteen and thirty years of age; they have perfect binocular fixation, but no binocular vision or sense of the "third dimension," they have never had the

slightest tendency to squint, yet the amblyopic eye follows the other in all parts of the field of fixation. I have tested these cases over and over again, and have invariably found a tendency to exophoria, or in other words, by the test in which I am said to be in error, the relative power of the externi was the same as that of the interni, if not slightly greater. Abduction was the same as adduction, and with the absence of the fusion power there was no tendency to turn the amblyopic eye inward.

Furthermore, I have never seen an explanation of the cause of squint in myopia when it is convergent. Surely "the position of rest" cannot cause it, nor the last clause of Donders' antithesis; but, on the other hand, a decided *weakness of the externi* may readily cause the eye to turn in under the stimulation for fixation at the near point.

Again, if the innervation, the position of rest, the hyperopia, or any other of the theories advanced is the cause of the strabismus, then why do we operate on the muscles by tenotomy or advancements that simply and only alter the relative power of the straight muscles of the eyes? Edward Jackson well says: "How will an operation influence the innervation of the muscles acting on the eyeball, or will it leave the innervation quite unaltered?" Does not this muscular theory apply forcibly to the action of the muscles in the vertical meridian, in which we have an upward or a downward strabismus?

Maddox says, "Non-paralytic squint, therefore concomitant, is purely due to excessive activity of the converging innervation," but I do not think his arguments prove it, as squint does not always disappear under the influence of an anesthetic, and "innervational habit" has nothing to do with physiological conditions.

I would also refer to the "natural cure of squint," in which you will have the history of convergence of one of the eyes for several years, until they have become about

thirty years old, when the squint disappears and they have perfect binocular fixation. This, as far as my observation goes, always occurs in the squint of the first class, or those with congenital amblyopia. Now examine these cases and you will find the amblyopia the same in reference to the vision, but if you carefully test the relative power of the lateral moving muscles by the prism test you will have abduction the same as adduction. Now what has become of the "innervation" or the "habit"? Has not the contracted internus lost its power after all these years and the eye found its natural position and follows the other?

I do not wish to present many illustrative cases nor many arguments in favor of this muscular theory, but I shall be pleased to see the case of squint of any kind that cannot be explained under the head of one of the two classes (always excepting any case of paretic squint), with an essential weakness of one or more of the muscles.

Finally, with these conclusions in reference to the theory of strabismus, granting that they are correct, the management of all our cases is clearly indicated, having determined the class to which the case may belong. It only requires the intelligent application of the treatment to end in success.

As many children are brought to us too young to be tested in reference to the visual powers, either by the use of the test-type or glasses, Worth has suggested a simple method that may give some very useful information as to the visual acuity. If the child is too young to know the letters or to count the fingers, Worth covers one eye with a bandage and then taking different sizes of white marbles or ivory balls — say from one-half inch in diameter to two inches — he rolls them on the floor away from the little patient, and after the marbles have passed about twenty feet the child is permitted to run after them. In this way, by the ability to find the marbles and the direction

the child will run we can easily judge the visual acuity and record it in the usual way. It will be best to test the fixing eye first.

Now what are the indications of the first class, or strabismus with congenital amblyopia? I do not believe that in any case or in any way we can restore binocular vision; but our aim must simply be to equalize the muscular balance and so attain a perfect cosmetic effect in order that the eye may keep its natural position with reference to the fixing eye. After an operation glasses may be ordered and used, if there are any indicatious for them, but all attempts to restore the vision will be useless.

Our treatment then becomes simply one of an operation to improve the cosmetic appearance, and that by one operation only, as *shortening of the weak muscle* and a complete tenotomy of the powerful one. If we put a "tuck" in the weak muscle, externus or internus, as the case may be, with the catgut suture, then do a complete tenotomy on the opposing muscle, and tie our suture so as to bring the eye into the primary position we will have corrected an ocular defect permanently, with a single operation. I have followed this procedure in my office and clinic for several years and have yet to record the first failure.

In this class and by this method we will not have to operate on the fixing eye — a great comfort to our patients — while the suture will be absorbed, so that we do not require a second operation for its removal.

These cases cannot be cured by the use of atropine and glasses, but must have an operation just as soon as the visual power can be tested, so that we may decide if one eye is amblyopic, and after that, order glasses if needed. Amblyopia is usually associated with hyperopia, but I do not think that the refractive condition is an essential factor in the causation of this class of strabismus.

In the treatment and technique of our second class of cases — and these are in the majority — we are now confronted with a far different problem, in which we must seek to restore binocular vision, perfect fixation, and the ultimate restoration of the vision in an eye that has from disuse partly lost its power to see.

Now we have two conditions to consider — primarily, an imbalance of the ocular muscles; and secondarily, a refractive error. These two conditions must be worked out with all the objective and subjective tests at our command before we take into consideration any operative interference. Here we may have all kinds of refractive conditions and all the different degrees of squint, not latent, but alternate, periodic, or fixed, from that of the slightest diplopia to a fixed squint, in which there is monocular vision with the fixing eye and generally reduced vision, with, perhaps, an eccentric fixation area in the other eye.

Hence, the exact and correct estimation of the refraction, even under the full effect of a mydriatic, is the first essential, as while I believe all these cases are associated with an imbalance of the ocular muscles, either those acting laterally or those acting vertically, the correction of the refractive error will in many cases so relieve the strain on the fixation power and *restore the guiding sensation* that the use of proper glasses will correct the tendency to a malposition of the visual lines and the squint disappear. Some writers claim twenty-five to thirty per cent. correction by glasses. My results fall far below this, possibly ten per cent. But this procedure will not succeed in all cases of the second class, and after the glasses have been adjusted the squint sometimes remains. We must then decide on an operation and look for our indications in the power of the opposing muscles.

Upward or downward squint will, as a rule, only require

a complete tenotomy on the most powerful muscle unless there is a decided weakness shown, when a shortening would be indicated; while in the lateral squints, particularly those of slight degree, we should first advance or shorten the weaker muscle or muscles, if indicated, and if the power of rotation inward is still too great in the field of fixation, we may safely do a complete tenotomy.

All cases of this second class must be carefully studied according to their degree and indications, but in *all* the correction of the refraction must *follow* the operation.

In the first class I do not think that the educational treatment will be of any service, but in the second class the education of the squinting eye to fix and appreciate the images will be of great service, and I might say that the object of this work is to prove that in the first class of cases it is useless to resort to any measures except operation, and in those of the second class we must resort to all reasonable measures, before deciding to operate. I have tried to show the indications for each procedure.

I do not think that special tables of all the cases of squint — both convergent and divergent — that I have records of will be of much service, but a résumé of the cases before mentioned may be helpful. The onset shows that they may occur at any age, from that of the first week in life (two cases), to that of twenty-eight years (one case). Dividing them into classes I have notes of

First class, $\frac{20}{100}$ or less in the squinting eye — 86.
Second class, $\frac{20}{70}$ or better in the squinting eye — 106, and 37 vision not recorded.
Glasses ordered in 48 cases, all second class, with correction of the squint in 16 cases — about ten per cent.
Five cases gave a history of operation before they were seen by me, with failure of result.

Operations. All cases operated upon in the office showed an excellent final result in cosmetic appearance,

but very few had binocular vision; while I have failed to find any material improvement in the vision of the cases which seem to have congenital amblyopia. The operation performed in my earlier cases was simple tenotomy — single or double — of which I note thirty-five with good final results, divergence following after some years, in two cases.

The operation for shortening with the catgut suture was performed sixty times, in all of which I had a good result, though many of them also had a tenotomy at the same time. I have had few, if any, accidents with my operations, in one case a small artery was cut, with free bleeding behind the eyeball, causing a slight exophthalmos. This was readily absorbed under a pressure bandage. In another an hæmophilic (I did not know of this beforehand), after shortening the internus I had bleeding from the wound, quite serious, for ten days. The constant efforts to stop the hemorrhage seemed to destroy the effects of the operation and the result was a failure to correct the divergence.

Fourteen cases are recorded as having been examined by the tropometer and all of these showed weakness of the externi in convergent squint, and of the interni in divergent squint, with decided improvement in the rotation of the eyes in the field of version after the operation.

Finally, I now consider that the best operation for the correction of squint is that of shortening both of the weak muscles in the second class, and then a careful tenotomy if needed; while in the first class a shortening of the weak muscle and tenotomy of the opponent on the amblyopic eye will be all that is necessary in the vast majority of cases.

Apparent Squint. A few words as to this condition will be necessary for the sake of completeness. As the term implies, this is not a true squint; in other words

there is no deviation or tendency to deviation of the visual lines but simply an apparent deviation of the optic axes with true fixation of the visual lines, and true binocular vision. This statement may seem somewhat paradoxical, but we must consider that we do not judge the position of the eyes by the visual lines, which we can not see but by the position of the optic axes, or, in better words, by the position of the center of the pupillary space. Now it has been well proven, by careful scientific examination, that the visual lines and the optic axes do not coincide but form a certain angle, the apex of which is really at the rotational point of the eye, but for all practical purposes we may consider that these lines will cross at the nodal point of the eye, situated at the posterior part of the lens. We have then two axes to consider in this subject: First, the visual line, drawn from the center of the object looked at, through the nodal point to the center of the macular region or yellow spot. This line fixes the vision on the object. Second, we have the optic axis, passing through the nodal point from the anterior to the posterior pole of the eye. These lines will form an angle at the nodal point, and this has been called the angle "alpha," or angle "a," and should be remembered in contradistinction to the visual angle. Landolt has designated this angle "a" as the angle "Kappa," for a full description of which I would refer the reader to his classical work on this subject. Now we find that in an examination this angle may be neutral, that is, the two axes will coincide or it may be *plus* or *positive*, when the optic axis passes to the outer or temporal side of the visual line, showing divergence, or it may be *minus* or *negative*, when the optic axis passes to the inner or nasal side of the visual line, showing an apparent convergence. From this it will be seen that according to the extent or size of this angle "a" we may

have an apparent deviation, judging from the position of the pupillary space, while there is present true fixation and no deviation of the visual lines.

Diagnosis. I give the method of Landolt for the measurement of this angle, from Norris and Oliver's System, Vol. iv, p. 46: "The affected eye being placed in the center of the perimeter, we cover the sound eye and have the patient fix the flame at the summit of the arc. In this case the visual line is directed toward the zero point o. If the visual line coincides with the pupillary axis the reflex of the flame would appear to be at the center of the pupil when we look at the observed eye from the summit of the arc. To find at once the direction and the degree of the angle we leave the flame immobile at the zero point and we move our eye along the arc until we have found the point at which it is necessary to view the observed eye in order that the reflex of the flame may appear at the center of the pupil. The corresponding degree on the arc then represents *twice* the angle." We can readily understand from this that the angle of incidence being equal to the angle of reflection the line of the optic axis must be on the arc, midway between the zero point and the degree on the arc.

Landolt adds: "It might seem that it would be simpler to have the patient fix the zero point of the arc, and carry the candle along the arc, in order to find at A. directly, this angle," and this method may be used in estimating small degrees of actual squint but the angle "a" is very small and we can estimate it much more readily by the first method. Hartridge states that the normal angle is about 5°, and Donders gives the angle in hyperopia as about 8°, and in myopia about 2°, but it is generally much less than this, except in cases where we have an apparent squint, when, of course, it is much larger. Maddox recommends the examination of the position of the optic axis

by the reflex from both cornea when illuminated by the ophthalmoscopic mirror. In this case the small images will be symetrically placed on the inner side of the cornea and if markedly misplaced will show real squint. This test may be useful in babies as a test for a very slight deviation. He further says, "The angle alpha is due to the fact that the fovea centralis does not lie exactly at the posterior pole of the eye, but is slightly below and to the

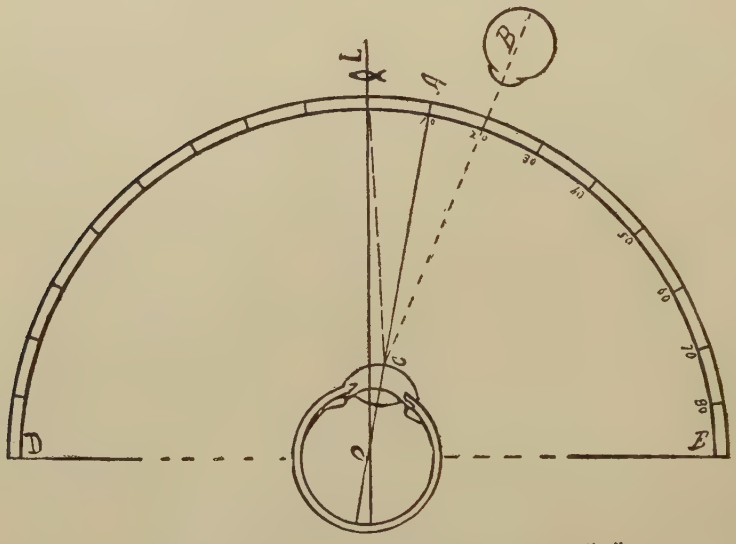


DIAGRAM SHOWING THE ESTIMATION OF ANGLE "A."

outer side, so that the visual line must cross the nodal point on the inner side of the anterior pole and when the eye is fixed and the image reflected from the cornea these images should lie to the inner side of the pupillary space. From this it is evident that a large angle "a" may diminish a convergent squint in hyperopia and a divergent squint in myopia, but these quantities are too small to be considered in real squint." This will be proved by the other test for the actual deviation, consequently this examination for apparent squint becomes useful when we

seem to have a deviation of the visual lines and yet, evidently, have perfect fixation. It goes without saying that no operation for this condition should be considered. I have seen these cases and the examination was very interesting, but they do not cause any asthenopic symptoms, and the slight defect in the cosmetic appearance of the case may be dismissed. Landolt claims that this condition should be considered in the treatment of true squint, but I have never found it necessary, and its influence is so slight in the cosmetic effect and not at all in the ultimate result that I have not suggested it in the treatment of the condition of heterophoria or that of heterotropia.

Amblyopia and the Restoration of Vision. As I read a recent article by Dr. A. E. Davis, in the *Post-Graduate*, March, 1900, on "Non-operative Treatment of Strabismus," in which he speaks, incidentally, of amblyopia, these words of Dr. Drew occurred to me: "A well-known author has declared that even the axioms of geometry would be disputed if men's passions were concerned with them, and so it seems." From Dr. Davis' article one would judge that there was no true congenital amblyopia. While the writer endeavors to convince us that amblyopia does not exist *per se*, and argues against it, at the same time he admits that it does exist in certain cases. To use his own argument,—"One case of this kind being more convincing than all the negative evidence against it,"—so if the writer has met with one case, as his paper clearly indicates, then what becomes of all his arguments?

Again, I will quote from the same paper: "If this amblyopia is congenital, that is, organic, the educative or non-operative treatment of strabismus will be of but little benefit, indeed; but, if acquired, that is, functional, then its possibilities are wide and the outcome hopeful." A postulate that I have advocated for several years,

particularly in my last paper on "The Theory of Squint,"† and also in one on "Crossed Eyes," read before the County Medical Society, May 28, 1894. Now, in the above proposition just quoted, the writer acknowledges the possible existence of congenital amblyopia, and in the next paragraph of his article proceeds to argue that this condition does not exist in squint of any variety. But if congenital amblyopia does not exist frequently, will some one explain to me,—Why do we see many cases of amblyopia in one eye that never had strabismus, yet presenting all the objective and subjective evidence of it?

Schweigger considers this congenital amblyopia, and all opthalmologists must have met with these cases. I have seen many of them in very intelligent people, in which the vision in one eye is found to be $\frac{20}{200}$ or less, who state that the vision has always been the same in that eye, as they have been tested, and worn glasses for many years. They seem to have perfect binocular fixation, yet the vision has always been sadly deficient in the one eye. However, there is no squint or family history to that effect, nor is there any objective evidence with the ophthalmoscope that one eye is not as good as the other eye, while both are generally found to be hyperopic of the same degree. Certainly this is pretty positive evidence of congenital amblyopia.

Then, if we acknowledge that congenital amblyopia does exist more or less, of what use will the educative treatment be? Is it not a waste of time and want of proper appreciation of the nature and the causes of squint? Furthermore, of what use will our glasses and other means be in the cure or correction of squint?

All these questions come rapidly to mind as one considers the probable and possible cure of squint, and at

† The *Ophthalmic Record*, September, 1899.

once bring to the physician's mind the question,—when the case is brought to him for examination and opinion,—Shall I operate or not?

My writings on this point have been plain and explicit, as I have divided my cases into the two classes: First, if amblyopia is present, operate at once, as all our educative treatment will do no good; on the other hand, if we have only amblyopia ex anopsia, then we will be fully justified in using all possible means to effect a cure before we proceed to change the balance of the muscular power by an operation.

In *Gould's Year Book* we find the statement that they have "never seen marked amblyopia, $\frac{20}{200}$ or less, changed by operation,"—testimony that can be established by nearly all oculists when they have tested their cases carefully, both before and after the operation for squint. I cannot recall a case at present that I considered at the time a true congenital amblyopia, in which the vision has improved. I consider all the cases of reported improvement in the vision those of simple amblyopia ex anopsia, the cases that I would place in my second class. In other words, simply cases of reduced vision due to suppression of the visual image from non-use of the retina. This is a physiological function of the retina that is simply in abeyance, and that may be again restored by constant use, particularly so, when positively demanded, as shown in the oft-quoted case of Dr. Johnson, of Paterson, N. J.

Hansell recognizes congenital amblyopia, and though he does not classify his cases, yet states, when the amblyopia is high, V.=20/200 or less, that the vision is not improved by an operation. Landolt offers the same testimony.

Moreover, another fact that I have advanced before in favor of congenital amblyopia, and one which seems to

me rather conclusive, is that the amblyopia does not increase after five or six years of age, the earliest age at which patients can be tested accurately. To give an example, say in a patient five years of age, the vision in the good eye is 20/20 and in the squinting eye is reduced to 20/200 or 1/5 (assuming, of course, that vision was 20/20 in each eye at birth); again, on testing this same patient at ten years of age or later in life, we find the vision in the squinting eye still 20/200 — that is, not further reduced after five more years. The question arises, why does not the amblyopia increase after the first five years of life if it is due to non-use, as the conditions are the same for the eye after the first five years as before?

"One swallow does not make a summer," and let us for a moment analyze one of these cases, particularly the case furnished in full as an illustration: In May, 1894, X. B. is four and one-half years old; now, periodic squint; two years more, squint now marked and constant, V.=20/100 in L. E. This is the first true test of vision. One year more, V.=20/50 with glasses; one year more, no squint; two years more, last examination, no squint with glasses on, V.=20/30. Now this constant squint is not cured, only corrected with glasses, but what of the restoration of vision? When the little fellow, just beginning to know his letters, not accustomed to use the left eye, was tested, V.=20/100, but as he grows older, in six years' time, with full correction, thereby improving his vision and so stimulating his fusion force to fuse the images, we have the vision improved to 20/30. I am sorry to say I must consider this only and simply a natural result, not a restoration of a physiological function or a psychical suppression of the retinal image, and surely not a very convincing argument for the total absence of congenital amblyopia in another case.

It is undoubtedly a very difficult matter to "draw the

line" between that of true amblyopia and that from non-use, hard to say positively just where one may begin and the other stop, or how much the vision may be reduced from constant disuse, due to one eye constantly turning inwards. For can we say how good the vision may be and yet indicate a true congenital amblyopia; but it seems to me that nearly all observers generally "draw the line" between 20/100 and 20/200, or less. If the vision of the squinting eye is 20/100 or better it is probably a case of reduced vision from non-use, and may be very materially improved by glasses and the educative treatment, with an operation if necessary; while, on the other hand, if the vision is found to be 20/200 or less in the squinting eye, then the prospect of any improvement in the vision will be very doubtful, if at all, though we may have binocular fixation, the visual lines parallel, and the squint apparently cured.

It is very evident from the above that I do not have much confidence in the restoration of vision in a true amblyopic eye, but it must be understood that this work represents my own personal experience and not the writing of others. I may meet some cases that will alter my present opinions but until I do so I must leave the reader to form his own conclusions. In this connection I wish to add the experience of one or two who have been very enthusiastic on this subject. In 1894 I received a report from my friend, Dr. Titcomb, of fourteen cases of convergent strabismus in children, in which he claimed some excellent results in the correction of squint with glasses, and in a certain proportion an improvement in the vision. His report is as follows:

Case	1	7	years	old.	Glasses	correct,	Hy.	V. = 15/30	each.
"	2	12	"	"	"	"	"	V. = 15/30	"
"	3	5	"	"	"	no use,	"	V. = 20/40	amblyopia.
"	4	6	"	"	"	correct,	"	V. = 20/40	"

Case	5	8	years	old.	Glasses correct,	Hy.	V. = 20/40 each.
"	6	6	"	"	"	"	V. = 20/30 "
"	7	7	"	"	"	"Ah.& Hy.	V. = 20/20 "
"	8	9	"	"	"	" "	V. = 20/50 "
"	9	8	"	"	"	"	V. = ?
"	10	4	"	"	"	"	V. = ?
"	11	6	"	"	"	"	V. = 20/50 "
"	12	6	"	"	No report,	"	V. = ?
"	13	3	"	"	No treatment,	?	V. = ?
"	14	4	"	"	Glasses correct,		V. = 20/40 "

This is practically the substance of his report and it shows a remarkable per cent. of correction for squint, in fact, almost one hundred per cent., and he was very much pleased with this excellent result. But, this year, 1903, Dr. Titcomb has sent me another report, in which he writes that he cannot trace all the cases, and "my conclusion is that the *majority* of cases of convergent, in children *with* or *without* amblyopia, but having marked refractive error, may be relieved by glasses alone." (The underscoring is my own). His 1903 report is as follows :

Case	1	No squint, V. imp. to 20/20 each eye.
"	2	" " with glasses.
"	3	Operation two years after first report.
"	4	Squint same, says does not wear glasses.
"	5	No squint, V. improves ?
"	6	Operation, after wearing glasses eight years.
"	7	No report.
"	8	" "
"	9	" "
"	10	No squint with glasses.
"	11	Result unsatisfactory ?
"	12	Operation, followed by marked divergence.
"	13	No report.
"	14	No squint with glasses.

These records are very interesting, but on what grounds does he base the assertion of "with or without amblyopia" when both of the amblyopic cases (Nos. 3 and 4) were fail-

ures. Furthermore, this second report only shows about thirty per cent. of successful cases with glasses, if we read it carefully, and even these cases seem to be selected ones. It is to be remembered that these cases were under observation several months, many of them fully under atropine and glasses, and yet in the final report only five show a good result. It would take some more convincing argument than the previous conclusions to change my opinions. We have also the report, published in the *N. Y. Medical Journal*, Jan. 2, 1897, by Dr. Conners. As I know he is familiar with my work I have great confidence in his examinations. He gives, in his report which I take the liberty to repeat, these cases:

Case 1.— Age 13. Convergent squint, 30°. V. R. fingers at 3 ft. L. E. 20/20. Operation and correction of the squint. Refraction + 3.50 cyl. 90°. By exercise and exclusion pad R. E. = 20/40. (What was the vision of R. E. with glasses before operation?)

Case 2.— Age 25. R. E. 4/200, L. E. 20/20. Convergent squint, 15°. Operation, corrects squint. After exercise, R. E. 20/100 with —7.50 D. (Did this case have amblyopia or myopia in the R. E?)

Case 3.— Convergent squint, 30°. R. E. 15/15. L. E. 10/200. Operation, tenotomy and shortening, corrects squint. Exercise and exclusion pad, 3 months. L. E. 15/40, with + 2. ⊃ + .50 cyl. 90°. (Again I must ask, what was the vision with glasses before the operation in the left eye? This is not amblyopia.)

Case 4.— Age 38. No squint. R. E. 15/200, with + 2.50 ⊃ + 2.75 cyl. 90°. L. E. 15/15, Hm. + 2. ⊃ + .75 cyl. 90°. Exercise and exclusion pad. R. E. 15/40, with correction. Reads Snellen No. 6. Treatment, strychnia and phosphoric acid.

This is the only case in this report that to my mind shows any improvement in an amblyopic eye, yet it does not seem conclusive. In the first three cases the doctor does not tell us what the vision was at first with a proper correction of the refraction, but in his final test she gives us the improvement *with the glasses*. Perhaps, had he fully corrected the refraction *before* the operations he

would have found the same improvement. I regret to say I would need some more positive evidence and a more full report before I could conclude that these cases present any serious evidence of the restoration of vision in what I consider a true amblyopic eye.

Finally, I report a case under my own care of improvement in the vision, in which the conclusions are obvious:

Irene D., age 7, 1897. Left eye turns in. Squint periodic. Hy. = 6. D. ord. + 4. D. each. 1898. V. = 20/70, with + 2,50 D. 1900. With correction, V. = R. E. 20/50, L. E. 20/70. Ord. wear glasses and cover R. E. five minutes daily. 1902. Has Hy. with Ah. axes to nose, with correction, R. E. 20/30, L. E. 20/50, no squint with glasses. 1903. Has stereoscopic vision and bar reading. V. with correction. R. E. 20/20. L. E. 20/40+. Trop. R. E. 25° up; 40° in; 50° out; L. E. up; 60° in; 30° out. Add. 30°, Abd. 14°.

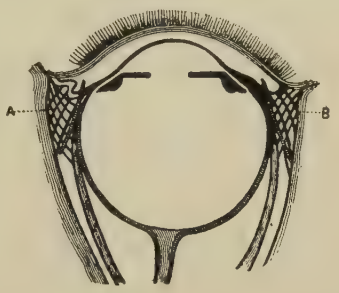
The fields of fusion and version show why this child had a tendency to squint and the continued improvement in the vision by the use of glasses shows the counteracting effect of the muscular imbalance, yet this is not a case of amblyopia but one of ex anopsia.

But why multiply argument on argument? The matter will still stand as it does to-day. Who is right? Time alone will tell. We may yet know the truth or the true cause as the science of ophthalmology advances, but even then I fear the axioms will be denied by some. I am ready to be convinced either way, but until then may I not conclude in the language of Dr. Drew: "If a thought offered or conclusion reached excites sufficient interest and reaction in your minds . . . . to call forth a free discussion, the best results hoped for will have been obtained."

Check Ligaments. The portion of the tissues that forms the sheath of the eyeball and the muscles anteriorly (tenons capsule), has an attachment all around the circumference of the orbits and in the region of the inter-

nal and external canthus becomes much thicker than in the other parts. This thicker portion is called the check ligament. They are attached to the orbit at the anterior part and the posterior portion is attached to the fascia and to the muscles by fibrous bands. These ligaments are supposed to hold the eyeball steady and to oppose an excessive action of the muscles when contracted.

Hansell and Reber has suggested that the attachments of these ligaments may have some remote cause, from their anomalous arrangement, in the failure to correct a squint by a free tenotomy. It is possible that these ligaments may have some influence in the final result of our squint operations, but in all my cases the rotation of the eye in the field of version has been so free that I do not think these ligaments have any influence. But, in this connection, I would refer the reader to those cases, not infrequently seen, called "Congenital absence of the outward movements of the eyes"—seeming convergent squint—where I think the check ligaments may have a very important influence, as in these cases there is a very decided limitation of the outward movement of the eye without any evidence or history of paralysis. I do not think this has been suggested before,—Leszynsky has reported one of these cases with this final conclusion: "Congenital arrest of development affecting the external recti muscles or possibly a faulty insertion of those muscles," but it seems to me that an abnormally developed check ligament at the internal canthus would more clearly produce the symptoms than if the fault was in the muscles or their insertions. I report a case that was examined by myself



(*a*) INTERNAL CHECK LIGAMENT.
(*b*) EXTERNAL CHECK LIGAMENT.

that seems to indicate the influence of these check ligaments on the rotation of the eye.

Miss A. R., age 13. Convergent squint, each eye, static condition, 15° inward. Noticed in first six months of life. Each eye will follow the test object inward but not outward beyond the first position; does not complain of diplopia; turning the head slightly to the right or left, V. = 20/15 in each eye. With red glass before one eye she has slight crossed diplopia. Refraction hyperopic, less than 1. D. Fields normal; convergence normal, and fusion near point seems at two inches with binocular fixation, but she does not have binocular vision; has no field of fusion; perimeter shows convergence of fifteen degrees with fixation generally in the right eye.

Tropometer.= R. E. 20° up; 50° down; 15° in; 0° out.

" = L. E. 30° up; 45° down; 35° in; 0° out.

This examination seems to show a certain limitation of the outward movements of the eye that may be due to a congenital shortening of the internal check ligament which restricts the outward movement to a point slightly inside of the first position, with fixation of one eye by rotation of the head.

CHAPTER VIII.

ILLUSTRATIVE CASES.

THIS series of cases is given in detail to show more fully the method of examination, the indications for the operation, and the final results. I have tried to illustrate in this way the various conditions of heterophoria and of heterotropia that may be met with in the usual office practice. All of them had the refraction carefully corrected by myself, or by others fully competent to do so, but wearing their full correction did not give them relief from their asthenopic symptoms. A careful comparison of the details of these examinations made before the operation and then afterwards was very instructive to me as I was interested in the binocular effect, that is to say, the effect on the muscles of the non-operated eye. In nearly all of these cases I could note the change that occurred and that seems to me a physiological process very difficult to explain unless we can see some psychological influence acting on the muscles at the same time.

CASE.— Paralysis of Convergence. J. D., age 45. Seen at clinic, 1902. Had an attack of hemiplegia, left side, says he is much better under treatment. Left leg only affected now. Complains of diplopia when on the street. Cannot read; V. = 20/20, each, and he seems to have binocular vision at twenty feet, but he cannot converge the eyes to a point nearer than one foot, when he has crossed diplopia. The eyes seem to fix at that point. Has Add. = 8°; Abd. 4°. Tropometer, 10° up; 60° down; 55° out; and 50° in. This examination shows that the associated movements of the eyes are normal; his field of fusion well balanced but small, and his field of version shows a tendency to kataphoria. It is evident in this case the cause of the trouble must be central.

CASE.— High Myopia with Hyperphoria. Mrs. H., under my care the past nine years. Has myopia of 18 D; V. R. = 20/30 V. L. = 10/200, due to large macular choroiditis. These changes at the macula have become perfectly quiet; has good fixation at twenty feet; right hyperphoria of 8°, with an exophoria of 4°, by Rod test; Add. 6°; Abd. 8°. In 1895 had tenotomy of R. Sup. and L. Ext. Decided improvement.

CASE.— Myopia with Exophoria. Mrs. R. H. P., age 38. V. R. E. = 20/40, L. E. = 20/15, with correction of the myopia and Am.; slight hyperphoria, = 1°; Exophoria = 10° Rod test. Supraduction, R. 3°; L. 4°. Add. 6°; Abd. 15°. Tropometer 25° up; 45° down; 45° in; 50° out, each eye. Operation: Tenotomy, both externi. This case has a long corneal curve, showing increasing myopia, but at the present time has had no symptoms of muscular asthenopia and illustrates the effect of correcting the lateral balance first.

CASE.— Myopia, with Exophoria and Kataphoria. Mrs. B. F. M. 1901. Cannot use the eyes and has various nervous symptoms. V.=20/15, with — 5. D. each. Add. 15°; Abd. 12°; Exophoria=5°; Oph. shows My. of 5. D. and no staphyloma, radius of cornea, =7.5mm. Tropometer, 10° up; 60° down; 40° in; 45° out. Divergence under cover test and at near point. Operation: Shortening of both Interni. 1903. Decided improvement in nervous and general condition. Add. 30°; Abd. 8°. Trop. 10° up; 60° down; 40° in; 40° out. The tropometer shows the weakness of both superior or the tendency of both visual lines to tend below the horizontal plane (Kataphoria). Lateral motion perfect, so I ord.— 5. D. ⌒ prisms 3/4° base up over each eye. A shortening of both superior would obviate the use of these prisms but as she has improved so much, no further interference is necessary.

CASE 2925. — Myopia with Exophoria. 1901. Mr. G. W., age 44. Dentist, cannot work. Refraction, My. w. Am. 90°; V.=20/30, each; Exophoria, 8°. Trop. 40° in; 45° out. Add. 20°; Abd. 12°. His excessive abduction indicates tenotomy and I did a partial tenotomy of the L. Externus, cutting all the upper and lower part of the tendon. 1902. Better, same operation on the R. Externus. He still shows some exophoria and I shorten the R. Internus. Slight hyperphoria. Result, complete relief. Add. 20°; Abd. 8°. Trop. 45° in; 45° out. An interesting feature of this case was the fusion near point, at three inches, while he had an exophoria of 8° at distance,

but I think this was due to the increased illumination and in this way a greater stimulation of the fusion force. It also shows the effect of these operations when this function is active, and Stevens' suggestion, that a tenotomy of the externus has much less effect on the action of rotation than that of a similar operation on the internus.

Case 594.— M. Myopia with Convergent Squint, First Class, and Amblyopia. Feb., 1903. Miss F. E., age 14. R. E. w.— 7. D.◯ — 1.50 cyl. ax. 180°=20/30. L. E. w.— 7.◯ — 2.50 cyl. ax.160°=20/100. Short radius of corneal curve. No changes at fundus. Onset, at birth. Excessive convergence. Here are all the essentials for divergent squint, high myopia and amblyopia, yet the eyes turn in. I can see no other reason or cause than the weakness of the externi. Operation: Shortening of the externus and tenotomy of the internus. Nov., 1903. V.=same. Trop. shows good field of version except weak inferior of the L. E.

Case 2593. — Myopia with Convergent Squint, Second Class. 1899. Mr. B R., age 25. Left eye turns in; no binocular vision. Refraction, My. 6. D. with Am. ax. 90°. V. R. =20/15; V. L. =20/30. Convergence, 40°. Operation: Shortening of the left externus and complete tenotomy of the left internus. 1902. Result, perfect. Add. 25°; Abd. 6°. Binocular vision, as shown by the stereoscope and bar reading. After the operation the Trop. shows, 30° up; 55° down; 55° in; 55° out, each eye. This case is very interesting from the high myopia with convergent squint completely corrected by operation on the squinting eye only, with perfect binocular vision. It must point to weak externi as the essential cause, as I cannot see what neuro-pathology can have to produce the squint, nor how the operation could have influenced the action of the innervation in the field of version.

Case 2918. — Convergent Squint, Second Class. Onset, late in life. 1901. Mr. S. G. A., age 32. Asthenopic symptoms several years, after an accident? The left eye turns in. V. =20/15. each. Refraction, Em. Careful testing shows hom. diplopia of 40°. Trop. 50° in; 30° out, each eye. Operation: Shortening of left externus, leaving hom. dip. 20°. Then tenotomy, left internus. Result, perfect. One year after, perfect binocular vision; no asthenopic symptoms. Esophoria,=1/2°. Add. 12°; Abd. 3°. Trop. 45° in; 45° out. This case shows many years of muscular asthenopia or esophoria, eventually, at the age of 30 changing to convergent squint (simply one of degree) and the examination of the field of version

shows that the accident was not the cause nor was it a case of the so-called "paresis of divergence."

CASE 2680. — Convergent Squint, Second Class. Master S. C. L, age 6. Onset, 3 years. L. E. turns in; V.=20 15, each. Refraction, Hy. w. Ah. 90°. Operation: Shortening of the L. Externus and tenotomy of the L. Internus. Result, perfect cosmetic; seems to have binocular vision at distance and good stereoscopic vision. Add. 20°; Abd. 15°? Tropometer, R. E. 30° up; 50° down; 50° in; 40, out. L. E. 40° up; 50 down; 60° in; 40° out. This shows slight hyper-esophoria still remaining. 1903. This boy has no squint and good binocular fixation.

CASE 2470. — Convergent Squint, First Class. Mr. J. E. B., age 37. 1899. R. E. turns in. Operation 16 years ago with no change in the visual line. V. R. =20/20; V. L.= fingers at two feet; Hm. 1. D., total Hy. 4. D. The left eye shows a high degree of astigmatism, but no improvement with cylindric glasses; Trop. R. E. 40° in; 40° out. L. E. 50° in; 35° out. This shows the fault to be principally in the left externus, so I shorten this muscle and cut the tendon of the internus. Result, 1903, four years after, perfect cosmetic. Says he has some crossed diplopia, due to eccentric fixation. V.= same as at first examination.

CASE 2464.—Convergent Squint, Second Class, corrected by glasses. Miss M. V. W., age 7. Squint alternating. V.=20/15 each, with + 2. ⌒ + 1. 90°. With this glass no convergence of the visual lines. Four years after, 1903, conditions the same. No squint with glasses, but the tendency to esophoria is the same, as Add. 15°; Abd. 1°, and Trop. R. E. 55° in; 48° out. L. E. 50° in; 45° out. This is a good illustration of those cases said to be cured with glasses. It will be noted that the muscles of abduction are not very weak in the field of version and so the increased visual power by the use of glasses and the relief of the strain on the accommodation, corrects the inward tendency but the primal cause remains.

CASE 1929. — Same. Convergent Squint, Second Class. Miss J. P. D., age 7. 1896. Onset, at first year of age. L. E. turns in. V. under atropine, with + 6.50 D. =R. E. 20/30; L. E. 20/50. Under atropine the squint stops, and afterwards she has Hm. 2.50 D. This glass ordered and the child has perfect fixation but no binocular vision. Ophthalmometer shows 2. D. of astigmatism not shown by retinoscopy nor will she accept cylindric glasses. 1901. Accepts cylindric glasses and with correction V. improves. R. E. =20/15. L. E. =

20/20. Still no binocular vision. 1903. Cover test, and with red and green glasses left eye turns in, showing esophoria 4°, but with glasses seems to have perfect fixation at twenty feet, also has stereoscopic vision but not bar-reading. Trop. R. E. 60° in; 50° out; L. E. 50° in; 50° out. At times her Add. shows 12° and Abd. 12°, not constant. This case is reported as one showing Donders' theory from the correction of accommodation and convergence and shows also the supposed restoration of vision, 20/50 to 20/20. Furthermore, this case may illustrate Worth's theory as the fusion force is very low, but is simply due to the high hyperopia. When we examine the rotation in the field of version we find the true cause of her tendency to squint held in check by the increased fusion force caused by the correction of the refraction by glasses. Still the prime fault remains.

Case. — Divergent Squint, First Class. 1899. Miss C. S., age 15. R. V.=20/15, Ah. .50 cyl. 90°. L. E. 20/200, imp. + 1.50 D. cyl. 90°. Operation: Shortening left internus and tenotomy left externus. Complete cosmetic result but no improvement in vision. Binocular fixation. Here we have divergent squint with hyperopic astigmatism. I can see no reason for this except a muscular anomaly.

Case 1272. — Divergent Squint with Amblyopia. Miss C., age 34. 1892. Onset, when baby. Occipital head pain. Refraction, Ah. 90°. R. V. =20/40. + L. V.=20/200. Glasses of no use. Crossed diplopia, 26°. Perimeter, left eye turns out 45°. Operation: Complete tenotomy of L. Ext. 1900. Eight years after, perfect cosmetic result. No binocular vision. Vision same as at first examination. This case only presents one condition to me, the very weak Int. more pronounced in the Amblyopic eye.

Case. — Divergent Squint, after an Operation. Miss A. O., age 16. 1903. States that I operated on the R. E. nine years ago, probably by simple free tenotomy; eyes did well for a time, then the R. E. turned out. It is now very prominent. This case shows the danger of too free a tenotomy on the Internus and the Trop. shows R. E. 20° in; 50° out. L. E. 50° in; 45° out. Under ether I tried to advance the Internus; it was found at the inner canthus and not attached to the sclera in any way; it was brought forward and attached to the conjunctiva and a tenotomy of the externus performed. The result showed slight outward squint. Eye less prominent and Trop. R. 40° in, 40° out.

Case 432 W.— Divergent Squint or Exophoria? Mr. F. W., age 38. Tendency of the eyes to diverge since a boy. Has good converging power. Dizzy spells. R. E. turns out almost constantly. R. V.=20/20, w.—.25 cyl. 45°.=20/20+. L. V. 20/40, w.—.75 = 20/20+. Refraction, slight myopia. Operation: Shortening of L. Internus and tenotomy of Ext. One year after, perfect result; V. improves to 20/15. Here is an exophoric condition slowly changing to a divergence with almost emmetropic refraction. It shows the close relation between heterophoria and squint and must have been due to the weakness of the Interni.

Case 1388.— Exophoria with Hyperopia and Amblyopia. Mrs. J. H., age 35, is a case of typical squint of the first class without any deviation of the visual lines. Has had constant asthenopic symptoms since a child. R. V.= 20/70, w.+ 3.50⌒+ 75 cyl. 90°= 20/40. L. V. 20/200, accepts + 4.50 D. Has worn these glasses constantly since 1901. Has exophoria of 6°. Add. 15°; Abd. 20°. Trop. R. E. 40° in; 50° out; L. E. 50° in; 45° out. Tenotomy Right Externus. 1903. Very much better. Trop. R. E. 45° in; 45° out; L. E. same. Add. 20°; Abd. 15°. It will be noted that in this case the eye turns out while she has all the conditions for convergent squint. From the examination of the duction I thought a tenotomy of the very strong externi would be the best. The final result and relief was very satisfactory.

Case 464 W.— Exophoria with Hyperopia. Mr. W. L., age 8. 1898. V.= 20/30, w.+ 1.50 D. = 20/15, each eye. Add. 6°; Abd. 8°. Glasses of no assistance. Operation: Shortening of Left Internus. 1901. At school, no asthenopia, Hm.+ 1. D. Add 15°; Abd. 8°. We may call this excess of divergence but the essential cause is weakness of the Interni as indicated by the operation.

Case.— Exophoria. 1900. Miss A. P., age 26. Pain in the head, back of the neck and shoulders, nausea and car-sickness. Had glasses. Refraction, Ah. ax. 90°. V. 20/15, each. Exophoria 8°. Add. 6°; Abd 8°; superduction 1°, each. Trop. 45° in; 50° out; Operation: Shortening Left Internus. Result, 1903, Add. 15°. Abd. 5°. Trop. 45° in; 40° out. Has had no symptoms of asthenopia since the operation.

Hyperphoria with Hypophoria. Mr. W. E. P., age 45. 1899. Refraction. Hy. with Ah. 90°. Add. 15°; Abd. 8°. R. Superduction, 8°. L. Subduction, 8°. Presbyopic. Operations: Tenotomy of R. Sup. and L. Inf. Result, superduction 4°, each eye, complete relief. It is

noticeable in this case how late in life the muscular symptoms began to cause the asthenopia. 1903. Has had no further trouble with the eyes.

CASE 2629. — Esophoria, improved with Prisms. Mr. H. B. A., age 29. Refraction, Ah. 180°, V.= 20/15 each ; Add. 40°; Abd. 6°. Trop. 50° in ; 40° out, each eye. This case shows a decided tendency to Esophoria, but as the outward rotation in the field of version seemed equal I ordered full correction of the refraction, combined with prisms 2° each, bases out over each eye. One year after he was very comfortable with these glasses but I think a shortening of an Externus would have been the best procedure. He declined to have an operation.

CASE 2621. — Esophoria. Miss B. H., age 30. 1899. Glasses by others, no relief. Refraction, Ah. 90°. V.= 20/15 each. Add. 20; Abd. 1°. Esophoria, = 5°. Operation: Shortening R. Externus. 1902, reports complete relief since operation.

CASE. — Esophoria. Constant pain in the head and cannot use the eyes for reading. Refraction, Ah. + 37 ax. 90 . V. = 20–15. each. Add. 30°, Abd. 5°. Rod, Esophoria, 12°. Trop. R. E. 52° in ; 55° out ; L. E. 62° in ; 45° out. Depending on the test with prisms and the tropometer I shorten the left externus. Operation stops all pain in the head. Now, Add. 25°, Abd. 8°. Trop. R. E. 50° in ; 50° out. L. E. 55° in ; 48° out. Esophoria, 10°. It will be noted in this case that the esophoria, as shown by the rod test remains the same almost as before the operation, showing that we cannot depend on an examination that deprives the eyes of the guiding sensation.

CASE 2832. — Esophoria, with Amblyopia. Mr. W. J. C., age 20. Refraction, Anisometropia. R. E. w.+ 1.50 ◯ + 1.50 cyl. 90°=20/15, L. E. w.+ 4. = 20/200. No improvement. The L. E. turns in under the cover test. Hom. diplopia, 6°. Add. 10°; Abd. 0°. Trop. 50° in ; 45° out ; L. E. 50° in ; 35° out. Operation : Shortening Left Externus. Result, perfect fixation. Abduction 2°. This case is on the border line of convergent squint of the first class, yet with the amblyopia still shows some fusion power.

CASE 1244. — Esophoria, with My. and Am. 1893. Mrs. C. M. J., age 36. Glasses 6 years, no relief. L. = 20/15, with correction, each eye. Add. 40°; Abd, 5°. Esophoria, 3°. Operation, tenotomy L. Internus. Some shock after operation. 1893. Much better.

1901. Complete relief. In this case we have an esophoria associated with myopia, not to be accounted for on the usual theories of squint.

Case 2065.— Esophoria, with Myopia. 1897. Mr. H. A., age 47, artist. Must close one eye to see well. My.=2. D. V.=20/15., with correction. Add. 30°; Abd. 3°. Operation: Shortening Left Externus. Afterwards, V. is steady, with binocular vision and fixation. Add. 24°; Abd. 6°. This case shows simple myopia and yet we have the tendency for the eyes to turn inward. Is it not due to weak externi?

Case 2920.— Esophoria, with Hyperphoria. 1901. Mr. W. E. S., age 38. Refraction, Am. 90 . Glasses some years, no relief. V. R.=20/15, L. 20/30, with correction. Add. 20°; Abd. 2°, Esophoria, 4°, L. Hyperphoria, 1°. R. Supraduction 0°, L. 4°. Trop. R. E. 25° up; 50° down; 55° in; 45° out. L. E. 30° up; 50° down; 50° in; 40° out. Operation: Shortening left externus and tenotomy left superior. 1902. Binocular vision, Add. 12°; Abd. 4°. Still has a slight hyperphoria. V. has improved, no asthenopia. Trop. 35° up; 50° down; 50° in; 50° out, each eye. This last examination shows a normal balance in the fields of fusion and of version.

Case 3006.— Amblyopia, no Squint. J. B., age 30. Perfect fixation, no bar reading. R. E. V.=20/200 not improved with glasses. L. E. V.=20/30, w.+.50 cyl. 180°=20/15, Esophoria, 3°, Add. 4°, Abd. 4°. Trop. 25° up; 50° in; 45° out. This case has all the essentials, as formerly advanced, of convergent squint, of the first class, yet the right eye does not turn in, nor has there been any change in the vision of that eye since birth. I think this must be congenital amblyopia and yet the appearance of the retina, with the ophthalmoscope was perfectly clear. It is further evident that the lateral balance of the eyes is almost normal and while the rod test shows esophoria the duction test shows exophoria and the tropometer shows normal movements in the field of version. In the field of fusion we find the reason why this case does not squint, as adduction is the same as abduction. Now I do not think that this case will ever squint, but, if with the above condition we associate weak externi, then the amblyopic eye will quickly turn in during childhood.

Case.— Anaphoria. Miss J. B. Asthenopia past two years. Glasses of no assistance. V.=20/15, each. Will not accept any glass. She says she always likes to look up and the eyes feel better when she does so, also tends to draw back the head. Add. 20°; Abd. 8°. Trop. 40° up;

50° down ; 55 in ; 50° out. Ordered prism 1° base down over each eye. (See also case of anaphoria reported with operation in the chapter on hyperphoria).

Case 3570.— Left Hyperphoria and Esophoria ? Mrs. D. C., age 25. Has had trouble with the eyes since she was ten years old. Has been under the care of many others, wearing glasses. When she came to my office she was wearing R. E. prism 7°, base in ; L. E. prism 4°, base down. Says these glasses gave her more comfort than others but she is not relieved. She is still very nervous, pain in the head and the back of the neck ; reading causes nausea and she becomes dizzy. R. E. V.=20/15, imp. w.+.50 cyl. 80°. L. E. V.=20/15, imp. w.+.50 cyl. 70°. Objective examination shows this glass to be correct and so ordered. The left eye appears to stand above the fellow eye, a slight vertical squint or left hyperphoria. Esophoria = 5°, hyperphoria = 4°, by the rod test. Prism test shows, R. E. 3°, L. E. 6°, superduction ; Add. 20° to 25°, Abd. 14°, each eye.

Tropometer = R. E. 30° up ; 60° down ; 50° ~~in~~ ; 55° out.
" = L. E. 35° up ; 50° down ; 65° in ; 40° out.

Operation : Shortening of the left inferior with the catgut suture Final examination. Result : Head feels much better in every way. Orthophoria by the rod test. Superduction 3°, each eye; Add. 20°, Abd. 10° each. Tropometer,= 30° up; 60° down; 55° in; 50° out, each eye.

Remarks. There are several points of interest in this case. The history of long continued muscular asthenopia, showing that the condition was partly congenital! The fact that she could wear the prism of 7°, over the internus was surprising when we note the field of version, but I think the most comfort she could have found from her glasses was due to the prism over the left eye. Her astigmatism, very unusual in the position of the axes, seems not to have been corrected. The Tropometer shows the vertical imbalance but also esophoria. Following the suggestions of others I operated on the left inferior and this seems to have corrected the lateral imbalance. It is evident that the fault was simply and only in the inferior of the left eye which, in my opinion, was congenitally weak.

Case.— Frontispiece: In considering the cause of squint some years ago I doubted if the illiterate races could have squint of the second class, that is, squint in which hyperopia might be the cause. It occurred to me that all squint of these ignorant people, who seldom use the visual power at the near point, must have amblyopia or squint of the first class, as hypermetropes, who seldom call into action the accommodation and convergence, could not have any tendency to squint unless there was some other cause. On a visit to Charleston, S. C., in the summer of 1902, Dr. Hallock, of that city gave me the photographs of the individual shown in the frontispiece of this book, stating that the boy had been operated on and that it was a complete failure. Now this case fully illustrates squint of the second class in an ignorant farm hand who has never used the eyes at the near point — except to look for a watermelon — and yet it is evident that the vision is normal in each eye with a decided squint of the alternating variety. Had this case been considered in reference to the muscular condition, in which the evidence of the weakness of the externi is apparent, and had a shortening or an advancement been performed on both externi, and then a tenotomy, if needed, his squint would have been corrected with probably binocular vision.

CHAPTER IX.

THE INDICATIONS FOR OPERATING IN LATENT OR FIXED SQUINT.

It has always seemed to me difficult to decide the question: Is it best to operate on the ocular muscles for heterophoria and heterotropia, and, if so, what is the best operation to perform? I think the decision as to the necessity for an operation is a very important one, and the determination of what operation to perform is decidedly more important. I do not think nor contend that all our indications point to one operation, but that all our operations must be modified or elaborated according to the results given to us by the tests of the relative power of the straight muscles of the eye. Yet, if we accept the proposition that all forms of heterophoria and heterotropia are due to a deficiency in power of some one or more muscles as the primal cause, then that deficiency should be carefully looked for and corrected as the first steps in our operative procedure. Hence, the suggestion for an operation, to my mind, is ever present in all these cases, even if they can be corrected by glasses, but as to the performance of an operation that can be left to the decision of the patient. My reasons for this is that all cases of heterotropia are simply heterophoria in greater or less degree, and the correction of that condition would obviate the use of glasses in many cases. I am well aware that this is an extreme and radical view of these conditions, but I am giving my own views and opinions only.

To return then to my first proposition, as to the necessity for an operation on these straight muscles, we

have two conditions which are to be considered: First, is there a want of balance in the relative power of these antagonistic muscles? Second, can the patient be relieved by any other means than an operation? To answer these questions we have first to determine what is the balance of power in these muscles? I have demonstrated that the normal position of the eyes was one in which both eyes will be directed straight forward to a point about fifteen degrees below the horizon and with the look in infinity. In this position I consider the normal eyes in a state of rest. Placing the eyeballs in this position we will find that they may be moved, in any possible direction, by either the single or combined action of the straight muscles; that they can be converged by the action of the interni; that they can be diverged by the action of the externi, when assisted by artificial means, and that the use and purposes of daily life require the eyeballs to turn downward much more than upward.

This being the natural action of these straight muscles, we can readily understand that though they can be antagonistic to each other, yet the requirements of life and work are such that one muscle must needs be stronger than the other, at the same time the eyeballs will be held in their proper positions by that exquisite retinal function, the *guiding sensation* or fusion power, which alone pertains to the eye. Then if it is necessary that one set of muscles should be stronger than another and considering the necessities of sight, near and far, we can see that the preponderance of power must exist in the interni; that we should find the next in power to be the externi; then the inferior, and lastly the superior, according to the relative need of action by these respective muscles.

The question has been raised, and I am sorry to say

often quoted, that the prism test will not give us the true power of the muscles, because while testing one eye there will be a corresponding action of the muscles of the other eye; as if, while we are testing one internus with the prism base out, the externus of the other eye will contract at the same time to overcome the supposed stimulation of both interni by the action of the prism. In some of my cases I have received answers from my patients that would indicate some unusual action of the muscles of the other eye, as they describe the position of the lights when diplopia is produced by prisms, but these cases are only "exceptions to the rule," and these seeming contradictions are not confirmed by the other objective tests. As a rule the uncovered eye does not move from the first position and this I have proved very frequently, in my office, with the assistance of an intelligent patient. I proceed as follows: By placing the prism with the base outward over one eye until the patient sees the two images of the light, I then place Snellen's test-card behind the candle and have the patient read the letters as seen by each eye. I find that the image seen by the uncovered eye will be clear and the letters that can be read will be equal to 20/15. This shows that the image formed by this eye must be on the macula, as the slightest deviation from this region will reduce the visual power. The image seen by the prism-covered eye will be very much reduced in visual acuity as now the image must be formed on the peripheral parts of the retina, as the eye is turned in under the stimulation of the guiding sensation. We have no evidence of any innervation of the muscles of the other eye, except the occasional cases I have mentioned above, but as the action of one eye may be perfectly independent of the other then, as a rule, the eye will not move from the first position except under the

stimulation of the fusion power when an image falls on the peripheral parts of the retina.

Now we make our crucial test for the relative power of these straight muscles by placing prisms in the trial frames before the eyes, with the bases first inward, then outward, then upward, and, lastly, downward, over each eye separately. This process is continued by employing stronger prisms each time, as long as the eyes will blend the images of the candle or other test object and the strongest prism that can be used in this way over each muscle after several trials will represent or indicate the muscular power of the eye to turn in a certain direction in the field of fusion.

Now, in reference to the manner of employing this test, we first ascertain the power of the externi, for I consider the abduction power of this muscle to be, *as a rule*, constant. We next test the interni, and here we should proceed very carefully, as this muscle may respond to our test, and by repeated trials we find an increase in power. To guard against this we should continue the trial on alternate days until there is no improvement. Some will rapidly respond, while others will remain the same as at first. Recording these results, we then proceed to find the power of the inferior, and, lastly, the superior. What should be the result of this examination?

Again, referring to my previous articles, we should find a certain proportionate balance, as follows: First, in reference to the lateral moving muscles, if we take the power of the externi as one, then we should find that of the interni to be two or three times stronger, or as one to two or three. As, for example, if the externi can overcome the deviation of a prism of 6°, then the interni should overcome one of 12° to 20°; likewise, in testing the power to move the eye up and down, we should find the inferior stronger than the superior. I am convinced it makes no

difference what may be the power of any individual muscle, provided we find the above-mentioned proportion, namely, the interni stronger than the externi, and the inferior than the superior.

I can hardly agree with Stevens that the externi must overcome a prism of 8° to be of normal power, as that would indicate the interni should evercome a prism of 30° degrees. I do not think it is important what may be the abducting power, provided the adducting power shall be found to be much greater. I make it a rule to test the lateral moving muscles in all my cases, and have yet to record a case where I have found the power of these muscles anywhere near that suggested by Stevens. I frequently find the externi as high as 8°, sometimes 15°, but the interni seldom goes above 30°. Having completed our test with the prisms and noted the indications, we now proceed to examine the field of version with the tropometer. If this indicates the same imbalance as the fusion test, we are then ready to decide the necessity and the method of our operation.

To read the indications rightly we must accept a normal rotation of the eyes in the fields of fusion and of fixation, as set forth in previous lectures and then to *compare* the results of our examination of the patient's ocular balance with that of the normal standard, being careful to particularly note the relation that one rotation bears to another in regard to the proportion.

In heterophoria, what are the indications and what is the operative procedure? What will our examination indicate? We may first decide the condition of heterophoria by the rod test or the phorometer, by which we can only decide the presence of esophoria, exophoria, and hyperphoria, and the degree. The next step is the prism test, which will indicate the location of the *weaker* muscle or muscles, in all cases, as we compare the

results with the normal balance and the relation one to the other. We then proceed to the examination with the tropometer, also comparing this with the normal rotation. This examination should confirm in every way the examination with the prisms and it will also indicate if we have the conditions of anaphoria, kataphoria, or a tendency to turn to the right or left. All these examinations being carefully and repeatedly made we are ready to select the best operative procedure.

Now we will meet cases, as we study the results of our examinations, that show an excess of power; in other words, some of the muscles seem to be too strong. This condition has been called an "excess of convergence," or "excess of divergence," as the case may be. If this increase or excess is shown to be much more than the normal rotation — it is frequently so in exophoria — we may decide on a partial or a complete tenotomy, according to the effect desired, as the first operative procedure before we attempt to increase the rotational power by a shortening. But excess must imply deficiency somewhere, and when we endeavor to estimate the effect of an operation we must bear in mind that the result of a partial tenotomy will be obviously less than a complete tenotomy if we wish to weaken the rotation of an eye, and that, if a strengthening operation is indicated, the shortening of the muscle will give us all the desired effect, as if needed it may be performed on both eyes.

A careful analysis of the results of these examinations, in almost every case, will show some weakness of one or more of the straight muscles of the eyes, and if so, the indications become clear, namely, to improve and to strengthen the function of those weak muscles. I know of no operation that will accomplish this object so "quickly, safely, and judiciously" as that of the shortening process to be described. This operation was devised,

practised, and demonstrated by myself before I had any knowledge that others had been working in the same lines, but after I had performed a number of these operations and published the results in the *Post-Graduate Journal*, July, 1895, I was informed by Dr. G. C. Savage that he had described a similar procedure in the *Ophthalmic Record*, March, 1893. In this editorial Savage describes his operation as follows: He uses two needles on a silk suture, and these needles are passed, one above and the other below, through the muscular tissue to the tendon and then tied. In the details of the operation, as I have always performed it, I use the twin hooks to hold the muscles and to expose its muscular tissue. The tendon and muscular tissue is completely dissected, and the use and the method of the introduction of the *catgut suture* obviates the necessity of a second operation for the removal of the suture. By this operation, when the muscle is innervated by the stimulation of the guiding sensation, the shortening of the length of the muscle must cause it to have an increased power to turn the eye. The operation is fully explained, and as it has been repeatedly performed by myself and others it may be said to have passed the experimental stage.

The Indications in Fixed Squint. For the past year, with the assistance and suggestions of Dr. E. M. Alger, I have been noting the effect of different operations on the eyes in squint, that is to say, what effect will a certain operation have on the rotation of an eyeball when performed. It has been my misfortune to see a very few cases that had been operated upon by myself for convergent squint some years ago. They came back to me with a divergent squint coming on some time after the first operation. It is to prevent this result that I have endeavored to perform my operations. At the present time I think that the following conclusions will be the

safest and best method of procedure to obtain a permanent effect and in many cases, possibly, binocular vision. It will be noted that I have divided my cases of fixed squint into two classes, and in each class I think we should adopt a somewhat different procedure. In squint of the first class, with amblyopia, I operate on the amblyopic eye only in this way: Place the catgut suture in position (see Operations) in the weak externus (or internus, if divergent squint), and before this suture is tied do a complete tenotomy on the opposing muscle. Now tie the suture, drawing forward the muscular tissue until the eye is in its proper position. It is always best to do this operation under cocaine, as we then can see the effect of the procedure as the operation is completed. In divergent squint a slight overcorrection will be advisable if it can be obtained. I have adopted the above procedure for several years and have failed to see the case in which I was not satisfied with the result. In these cases I can only expect to get a good cosmetic effect as I have no confidence in the restoration of vision in a congenitally amblyopic eye, and consequently do not make any effort to get binocular vision in the after treatment of these cases. The refraction should always be corrected.

In the second class of cases, those with good vision in each eye or only amblyopia ex-anopsia, I now prefer to do the shortening operation on both externi, in convergent squint, on both interni, in divergent squint, and then to perform a very careful tenotomy on the opposing muscle or muscles, if needed, not lacerating the capsule of Tenon more than is absolutely necessary to put the eyes in the first position. This procedure, followed by suitable glasses to correct any refractive error, suitable exercise with the prisms, and other methods, should correct the squint, and, in many cases, result in that much to be desired condition, binocular vision and fixation, the ideal

eyes. I am well satisfied that in all cases of vertical squint, hyperphoria, the shortening operation is always indicated.

I would refer the reader to the chapter on Illustrative Cases for the practical application of all these indications, where we have the results of the examination before the operation, the operations performed and the attainment of the *normal* balance as the final result. Hence, we always have the following object in view, in the treatment of all cases of squint, either fixed or latent. In heterophoria, to attain the normal balance of duction and version, and in heterotropia, fixation, and binocular vision.

CHAPTER X.

OPERATIONS.

"The only type of heterophoric asthenopia that can legitimately admit of operative interference is the idiopathic." — "The fault must be located in the part upon which the operation is done." — "The fault should not be one of improper innervation from a central or peripheral cause." — "It is a local, physical deformity, with an imperfect and disturbing physiological result."—"Anatomic readjustment of improperly placed muscular tissue and tendon is the only legitimate result." — C. A. Oliver, M. D., in *Norris and Oliver*, Vol. IV., p. 465.

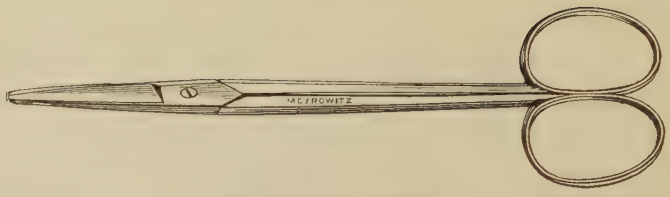
When we come to consider the operations on the eyeball for the correction of either latent or fixed squint, there are but few anatomical points to remember. Of these, the most important, and one that may possess some complications in the course of our operation, is that of the insertion of the tendons of the ocular muscles into the sclera. In my description of the movements of the eye I have stated the positions in which these insertions of the tendons will be found. They form somewhat of a spiral, if we take the insertion of the internus as being nearest to the limbus of the cornea. Then somewhat farther away we will find the insertion of the inferior, and that of the superior placed much farther from the edge of the cornea than all the others. These insertions become very important as they bear a very positive relation to an operation and form fixed points that should be fully appreciated, even before the conjunctiva is cut, that they may be quickly found when the incisions are made.

The size of the tendon should also be remembered, as we will find the tendon of the internus much larger than that of the other muscles. The tendon of the externus

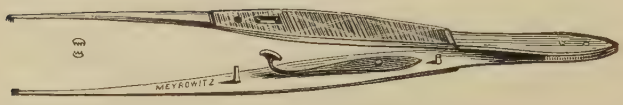
may be found very loose, as it does not seem to grasp the eyeball in the same manner as other muscles.

Asepsis. In reference to this subject, I think too much stress has been placed upon it in reference to operations on the ocular muscles. The field of operation certainly must be in an almost aseptic condition all the time, unless there is some pre-existing disease of the conjunctiva, and during the healing process we have the wound constantly bathed in the secretions of the lachrymal gland as they pass over the conjunctival sac to the puncta. Hence the only asepsis that we need is perfect cleanliness of the eyes and the hands of the operator. Thorough washing of the hands and sterilization of the instruments is the first requisite, and just before the operation the lids and conjunctival sac should be washed out with a solution of boracic acid or boiled water. With these preparations we are ready for our operation.

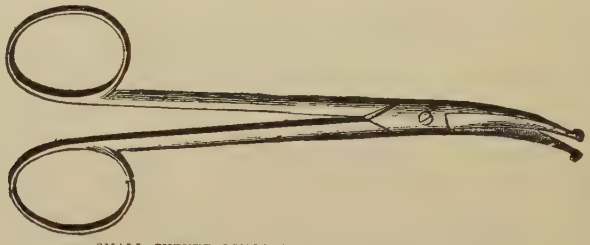
In the description of these operations I shall give them as they are performed by myself, while to those who may wish to follow other methods or other operations, I would refer to the writers on this subject, particularly to the article by Knapp, in *Norris and Oliver's System*, Vol. III. In these operations I describe those of cutting the tendons first, though, as I study the effects of the operations on the eyeball for any of the degrees of squint, I should put that of strengthening the muscle in the first place, for the reason that I believe the essential cause of all squint is a weakness of some one or more of the muscles of the eyeball. If that proposition be true, then it seems reasonable that our first operative procedure should be a strengthening one, to be followed by a weakening or tenotomy of the opposing muscle, if needed. In all my operations on the muscles of the eye I prefer the local use of cocaine in 4 per cent solution,—a few drops of this placed in the conjunctival sac before the operation being all that is



SMALL STRAIGHT SCISSORS.



FIXATION FORCEPS.



SMALL CURVED SCISSORS.

CAPSULE — STERILIZED CATGUT

SMALL AND LARGE STRABISMUS HOOKS.

TWIN STRABISMUS HOOKS.

NEEDLE HOLDER.

INSTRUMENTS NEEDED FOR THESE OPERATIONS.

needed to make the operation almost painless. I have never experienced any ill effect from the use of this drug. Before the operation I put one drop Sol. Adrenalin Chlor. 1 : 1000, on the conjunctiva. If an anæsthetic is desired for these operations, I prefer the nitrous oxide, except with children, when chloroform or ether may be used.

Partial Tenotomy. The object of this operation, it seems to me, is to produce a very slight effect on the rotation of the eye. We proceed as follows:

The eye being prepared as above, the speculum is introduced first under the upper lid, then the lower, and the eyelids separated only sufficiently to render the field of operation clear. The conjunctiva is now grasped with a pair of fixation forceps, exactly over the insertion of the tendon, raised up slightly from the sclera, and a small incision made. One blade of the scissors is then carried beneath the conjunctiva and the incision is enlarged in the direction of the muscle, horizontally if we are operating on the lateral muscles. The capsule of the tendon is now opened by a small incision into Tenon's capsule, and a small strabismus hook is passed beneath the tendon. Raising the tendon slightly on the hook, I now cut the fibres on both sides, as the upper and lower ones on the internus, by a number of small cuts, until the center of the tendon is reached. At this point I leave a small portion, about the size of a large thread attached, to the sclera. The hook is now removed, the conjunctiva smoothed back into its place, the speculum removed, and the eye washed with a solution of boracic acid.

Complete Tenotomy. In this procedure we obtain a greater effect on the motility of the eye, according to the extent of our separation of the capsule from the sclera. The steps of the operation are the same as those for a partial tenotomy, but the tendon is cut completely from the sclera, always commencing at the point of the hook

so as to cut all the fibres. If a decided effect is wanted, the expansions of the tendon are taken up on the hook, both above and below, on the lateral muscles, and completely severed from the sclera.

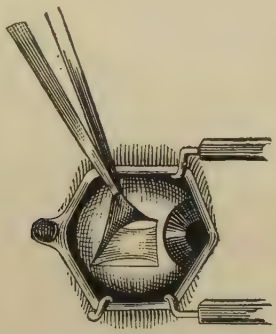
These operations require very little after treatment. For the first twenty-four hours, cold applications are very grateful, and after that time a lotion of boracic acid is all that is needed. The effect, which will be shown when the eyes are tested, is about 5 to 15 degrees in heterophoria. In squint, a decided change in the position of the visual line. This must vary according to the fusion force of the eye and the degree of the squint.

SHORTENING OF THE OCULAR MUSCLES.

The operation for advancement of the ocular muscles has been in use by ophthalmic surgeons for many years, and has been changed and modified by many, but as it is performed at the present time it is a delicate and extensive operation in which an assistant is required. Furthermore, the subsequent removal of the sutures is almost a second operation. I believe that owing to these conditions the operation is seldom performed unless it is absolutely necessary, as in cases of extreme divergent squint, paresis or paralysis of the ocular muscles. For the past seven years I have successfully attempted a different method to attain the same object, provided the result desired is not too great. I think that I have succeeded in devising a method of shortening the ocular muscles that is easy to perform, that does not require an assistant, or the removal of the suture, and that, moreover, produces an entirely satisfactory result.

I will describe the procedure and also the instrument that I find very useful and of great assistance while passing the suture. This instrument was made for me by Mr. E. B. Meyrowitz of this city.

When the eye is prepared for the operation and the specula inserted, the conjunctiva is grasped, not over the center of the tendon as in tenotomy, but over the lower or upper part of the insertion, raised up, and a small opening made. From this point I make a lateral incision, and then a vertical one forming an L. This is raised up very carefully and dissected from the sub-conjunctival tissue and laid back. We now grasp the capsule with the forceps at the lower part of the insertion of the tendon, a small opening is again made, and the hook passed beneath until its point protrudes on the opposite side. Another hook is now passed beneath the tendon in an opposite direction. These two hooks are now drawn apart, in this way exposing the tendon and muscle.



EXPOSURE OF THE MUSCLE, AFTER HANSEL AND REBER.

The twin strabismus hooks are now inserted under the muscle between the other hooks, which are then removed and the twin hooks allowed to separate from the pressure of the spring. If they do not separate sufficiently, or as far as I wish to shorten the muscle — and I judge this procedure by the indications, as to the amount of shortening necessary — I then place the blades of a pair of scissors between the hooks and open them forcibly until I get the desired result.

TWIN STRABISMUS HOOKS.

This instrument will stay in position without holding, and we are now ready for the insertion of the catgut suture. This is always used in the operation, as a thread requires almost a second operation to remove it. I use

CATGUT SUTURE IN CAPSULE.

a very small Hagedorn needle with a very large eye, so that it can be easily threaded. The catgut — sizes 0 or 00 — is used from a glass capsule holding sufficient for one operation.

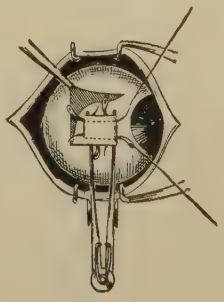


DIAGRAM OF NEEDLES, SUTURE AND HOOKS IN POSITION.

Armed with the needle and suture, we are now ready to pass it in the following manner: using the insertion of the tendon as a fixed point, the needle is passed through its upper or lower edge, including part of the sclera if possible, and going beneath the blade of the twin-hooks. We now carry the needle backward beneath the other hook and pass it through the muscular part from *within*

outward. Carrying the suture now across the muscle, we again pass the needle through the muscular part at a point opposite the last puncture, going from *without inward.* The suture is drawn down close to the muscle and the needle is now carried back to the tendon, passed beneath it at a point opposite to the first insertion, then through the tendon, close to the sclera. As the suture is drawn tight, it will now present the form of a parallelogram. (see diagram.)

The twin hooks are now removed and the two ends of the suture are carefully and evenly tied with a surgeon's knot over our fixed point, the insertion of the tendon. As it is drawn tight, we see the knuckle or tuck of the muscle forming as the muscular tissue is drawn forward. After the suture is securely tied, the ends are cut off and the flap of conjunctiva is drawn back into place over the tuck in the tendon, then the specula is removed. This flap does not need a suture to keep it in place, unless there should be some decided opening of the wound, when it may be readily closed with a small superficial suture.

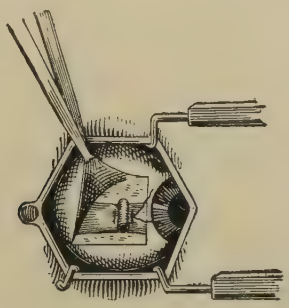


DIAGRAM OF SUTURE TIED, SHOWING TUCK,
AFTER HANSELL AND REBER.

The after treatment is the same as that of tenotomy, and at no time do I close the eye with a bandage, except in an office operation, and then only when they are going

home. I prefer this open treatment after the operation because it seems to me better that the eyes should be used as the healing process takes place, so that we can keep the fusion force constantly active. The results of this operation will be found to show about 10 to 15 degrees correction of the visual line in squint and a decided improvement in the fields of version and fusion in heterophoria.

I would state that I was the first to use the catgut suture to take a tuck in these muscles and allow it to be absorbed, therefore requiring no further interference with the eye. I have found this method very useful in convergent squint, in divergent squint, in paralysis, and particularly in insufficiency of the ocular muscles, where the indications are to strengthen the weaker muscles instead of, as in the old method, weakening the stronger muscles by a partial or complete tenotomy. Savage published the first account of this method of shortening an ocular muscle in 1893 — using a black silk suture to form the tuck.

I give the records of twenty operations performed by this method, and in all, the results, for the relief of the symptoms or the correction of the squint, have been very gratifying. In only one case has there been any failure of rapid and complete healing of the tissues and in this case I had a small stitch abscess due to a fault in the catgut used. The following twenty operations will illustrate the conditions in which I think the use of the suture is indicated :

For convergent and divergent squint . .	12 cases
For paresis or paralysis	1 case
For heterophoria, all forms	7 cases
Total	20 cases

These cases are not included in those of special deviations.

Case I. — Paresis of the Left Externus. F. A. F., age 30. Homonymous Diplopia. Operation: Shortening with catgut suture, 1894 Result, complete relief of the diplopia.

Case II. — Weakness of the Externi, Esophoria. Mrs. H. T. N., age 38. Pain in the head extending down the spine ; she cannot use the eyes ; when reading pain is much worse. Refraction examined under atropine and glasses ordered. After one month's trial they do not relieve the pain. Repeated examination shows a want of balance in the eyes ; Add. 20° ; abd. 0°. Esophoria = 6°. Jan., 1896, — catgut suture in left externus. Result, Add. 20°, abd. 6°. She was advised by another oculist not to have an operation. 1903. I have met this lady and she states that she has had no further trouble with the eyes.

Case III. — E. A. W., age 23. Sent to me by Dr. Carter. Headaches frontal, extending backward. Manifest hyperopia, +.50 D. with V. = 20/20, each; Add. 16° ; abd. 0°. May, 1896, catgut suture in the left externus. Result, cessation of the headaches and now Add. 16° ; abd. 4°. It was in this case that the slight infection of the wound occurred.

Case IV. — Weakness of the Interni, Exophoria. Miss A. S., aged 30. Headaches, frontal and occipital. Refraction, compound myopic astigmatism axes toward the temples, corrected with glasses, but after four months' trial no relief. Has crossed diplopia ; and by prism test, Add. 2° ; abd. 10°. In 1894–95, I did the shortening operation on both interni and a partial tenotomy on both externi. Result, patient was much better. Add. 16° ; abd. 8°, but she was not completely relieved from the asthenopic symptoms. It will be noted in this case, that the power of the externi, in the field of fusion is still too great.

Case V. — Mr. C. J. S., aged 32. Has not used the eyes for working or for reading during the past three years ; he is neurasthenic ; complains of drawing sensation in the back of the head and neck. This is a common symptom in these cases of heterophoria. This man has myopic astigmatism that had been fully corrected by glasses but they do not give him any relief. Crossed diplopia ; by prism test 4°, and at times has double vision. Add. 12°, changing by repeated trials ; abd. 10° ; Nov. 1, 1895, catgut suture in right internus. Two weeks after, Add. 30° ; abd. 8°. Two months after operation reports by letter that he is back at work, bookkeeping, and feels better than he has for several years.

Case VI. — Miss S. J., aged 25 years. Is very dizzy, and has had double vision for past six months. V. = $\frac{20}{15}$, — Hm. 50 D., Add 3° ; Abd. 5°. Catgut suture in right internus. Two months after, all symptoms disappeared and Add. 12° ; Abd. 5°. To use +. 50 D. glass for reading. I saw this lady fifteen months after the opera-

tion, and on examination the lateral balance of the muscles was Add. 16° ; Abd. 5°.

CASE VII. — Mrs. G. H., aged 49 years. Headaches, frontal, and has dizzy spells. V. = $\frac{20}{20}$ — w. + 1 D., cyl. ax. 90° = $\frac{20}{15}$ —. Add. 6° ; Abd. 6°. Catgut suture in left internus. Three months after, reports much better. Add. 12° ; Abd. 6°.

Convergent Concomitant Squint, first class, seven cases.

CASE VIII. — W. G., aged 20 years. R. V. = $\frac{20}{20}$ + ; L. V. = fingers at two feet. December, 1895. Tenotomy of left internus. Immediate result good, but squint returned. January, 1896. Catgut suture in left externus. Two months after, perfect cosmetic effect ; no change in vision.

CASE IX. — D. B., aged 17 years. R. V. = $\frac{20}{20}$; L. V. = fingers at ten feet. Has compound hyperopic astigmatism. September, 1895. Catgut suture in left externus and tenotomy of left internus. Perfect cosmetic result. Ordered + 2 D., cyl. ax. 90°.

CASE X. — S. D., aged 5 years ; glasses for two years, correcting refraction of + 1 D. in each eye, but no improvement in squint. Operation at hospital under ether. May, 1896. — Catgut suture in left externus and tenotomy of left internus. Perfect cosmetic result.

CASE XI. — Mrs. L. B., aged 31 years, Liberty, N. Y. R. V. = $\frac{5}{200}$; L. V. = $\frac{20}{30}$ w. + $\frac{1}{13}$ ⌒ + $\frac{1}{48}$, cyl. ax. 180° = $\frac{20}{20}$. Put catgut suture in right externus with perfect cosmetic result.

CASE XII.—E. A. P., aged 8 years. This boy has slight nystagmus in both eyes with the squint. R. V. = $\frac{20}{100}$, Hm. 2 D. ; L. V (?) ; oph. shows Hy. 6 D. May, 1895. Under ether and assisted by Dr. Coffin, I put catgut suture in left externus and did a complete tenotomy of left internus. Four months after, perfect cosmetic result. R. V. = $\frac{20}{100}$; L. V. = fingers at 4 ft. ; no nystagmus. Eight months after, same vision.

CASE XIII.— E. W., aged 23 years. R. V. = $\frac{20}{40}$, Hm. 1 D. ; L. V. = $\frac{10}{200}$, Hm. 3 D. ; opthalmoscope same degree of hyperopia. March, 1895. Catgut suture in left externus and tenotomy of both interni. Perfect cosmetic result.

CASE XIV. — F. L., aged 13 years. This young girl had hysterical amblyopia associated with her convergent squint. Refraction, compound hyperopic astigmatism. R. V. = shadows, L. V. = $\frac{20}{200}$. February, 1896. Catgut suture in left externus while under ether.

One month after, no squint and vision slowly returning. R. V. counts fingers, L. V. = $\frac{20}{70}$.

Divergent Concomitant Squint, first class, two cases.

CASE XV. — S. D., aged 15 years. R. V. = $\frac{20}{200}$; L. V. = $\frac{20}{20}$ —; can fix with both eyes at near point, but at distant vision right eye turns outward. March, 1896. Catgut suture in right internus. Has hyperopic astigmatism, and I ordered + .50 D., cyl. ax. 90° for each eye. Result perfect.

CASE XVI. — A. C., aged 15 years. Divergence of right eye since childhood. R. V. = $\frac{6}{200}$; L. V. = $\frac{20}{15}$. Refraction R. = compound myopic astigmatism; L. = Hm. May, 1896. Catgut suture in right internus and tenotomy of the externus. Perfect cosmetic.

Convergent Concomitant Squint, second class, three cases.

CASE XVII. — G. F., aged 7 years. Alternating squint. Hyperopia, 3 D. Atropine and glasses tried for three months; no result. January, 1896. Catgut suture in the left externus and tenotomy of the internus. Six weeks later ordered + 2. D. each eye. Result perfect.

CASE XVIII. — H. H., aged 13 years. Right eye turns inward. Esophoria, or slight convergent squint. R. E. V. = 20/200. w. — 4. D. = 20/70.; L. E. V. = 20/70. w. — .50 D. = 20/40. Under atropine R. E. = 20/200, w. — 1.50 D. = 20/70; L. V. = 20/70, w. — 50 ⌒ —.50 cyl. ax. 30° = 20/40 +. These glasses do not correct the squint or change it in any way, so I did the operation of shortening the right externus. The result was perfect.

CASE XIX. — D. O. C., aged 28. Convergent squint since childhood. R. V. = 20/50, w. + .50 cyl. ax. 90° = 20/40; L. V. = 20/20. improved w. + .50 cyl. ax. 90°. August, 1895. I did a complete tenotomy of the right internus and the next day put in the catgut suture in the left externus. In this case the result was excellent as we had binocular vision.

In all these cases, from my private practice, in which the suture was inserted twenty times, the relief of the muscular asthenopia, the paresis and the squint has been very satisfactory. In the asthenopic cases we have immediate relief and gradual improvement; in paresis the diplopia disappeared and in squint the cosmetic effect was all that could be desired. In all the healing was

clean and rapid with only a slight œdema of the ocular conjunctiva for a few days. In all my cases since these were reported I have not had any trouble with the healing of the wound and the absorption of the suture. I have had Meyrowitz prepare the sterilized catgut for me and put it in small capsules, each containing a sufficient quantity for one operation ; for we can only depend upon it being readily absorbed when it is perfectly sterilized.

In concomitant squint, either convergent or divergent, with amblyopia, I put in the suture and then cut the opposing muscle before the suture is tied, thereby completing the operation and placing the eye in its proper position with but one operation.

The suture can be easily applied under the anesthetic effect of cocaine, except in young children, when I prefer to do it while they are under ether.

Fifty years ago, before Donders' made his great discovery that asthenopia was due to hyperopia, the surgeons of that day frequently performed tenotomy of the lateral muscles for the relief of asthenopia, and in many cases gave relief ; but Donders' discovery seems to have changed the opinions of the ophthalmic surgeons, and we now depend on glasses for relief in all cases. But modern methods of investigation have proved that while many patients are relieved by glasses, still there are those which we meet with very frequently that do not derive the expected relief from their glasses ; and when we do find a decided want of balance in the power of the muscles, compared with that of the normal proportion, as stated in a paper by myself in the *Medical Record* of July 21, 1894, we are then justified in an operative procedure which will relieve the asthenopia.

In cases of muscular asthenopia I depend entirely upon the prism tests and the findings of the tropometer for indications in the use of the suture, for we should not

deprive the eyes of their most useful function, the guiding sensation or fusion power.

There has been so much opposition in the profession to the partial and graduated tenotomies, and still further so many failures, that some, I think, have perhaps ceased to operate; but I do not believe the same objections can hold in the case of this procedure. It can be applied to so many cases of insufficiency of the straight muscles without the least danger of overcorrection or producing squint, that I offer it to the profession for a fair and complete trial.

Advancement of an Ocular Muscle. — I think the results of this procedure are greater than that of other operations, and it is generally indicated in extreme divergent squint. The first steps of the operation are similar to those of shortening, until the muscle is exposed on the two hooks. Then I prefer to follow the suggestion of Prince, whose operation is called "the single suture" or "pulley" method. The operation requires great care and attention to detail, but the results are very satisfactory and the method removes that possibility of producing torsion by tying one suture tighter than the other as in the old methods, when two or more sutures are used. In performing this operation, I have exposed the muscle to be advanced on the two strabismus hooks, as in the shortening operation. While these two hooks are held by an assistant, we take a black silk suture armed with two small needles and pass one through the upper part of the muscular tissue, and the other below at an opposite point from without inward, and as far backward in the muscular tissue as needed. This forms a loop, which with the two ends are now taken in one hand, the hooks removed, and the tendon completely severed from its attachment. The loop is now laid aside and the upper needle is passed deeply into the sub-conjunctival tissue

beneath the conjunctiva until it emerges above the cornea about three or four millimeters in the vertical meridian of the eye. The lower needle is now passed below in the same way, coming out at an opposite point below the cornea. We may now take either needle and carry it back through the loop and then this loop is drawn down tightly over the thread. Now carry the suture back towards the other end, and as these are tied it will be readily seen that the cut end of the muscle and tendon must advance toward the edge of the cornea. An assistant may rotate the eyeball in an opposite direction at the same time, so as not to put too much strain on the tissues as the suture is tied. Any overlapping of the conjunctiva on the cornea will disappear when the sutures are removed. By passing the suture in this continuous manner through the loop or pulley, the movements of the eye will soon adjust the tension above and below, and so prevent torsion on the optic axis. The effects of this operation is very decided in the change of the position of the visual line. In very marked divergent squint, this operation should be performed in all cases, and may be done on both interni at one sitting. Landolt recommends this operation in all cases of squint, and it has also been warmly advocated by Wooten of this city, who reports some excellent results. I prefer to use it only in cases of excessive squint. The effects of this operation will show from 20° to 25° in the change of visual line.

CHAPTER XI.

AFTER TREATMENT OF SQUINT.

The suitable procedure with our cases after the operation has been performed will depend on the previous existing condition, that is to say, whether our case has been one of fixed or latent squint. In fixed squint, when the eyes are examined after the operation, if there has been any over-effect produced as a slight divergence, then I keep the eyes open and if a shortening or advancement has been made we may loosen the suture if possible. Conversely, if we find that the visual lines still turn inward slightly it is best to use the solution of Atropine (grs. 2 to the ounce), instilled into both eyes so as to stop all action of the accommodation and the refraction must be fully corrected with glasses. As I do not cover the operated eye when protected in the house, this procedure can be instituted at the first examination. If our operation for heterotropia has been successful as far as the position of the visual lines is concerned and the eyes appear to be in the first position when fixed on infinity, then the after-treatment is the same as follows in the cases of heterophoria, that is to say, the effort to improve the fusion force and to establish binocular vision.

I consider the treatment of these cases, for the correction of muscular asthenopia, before we have attempted the correction of any case of heterophoria by an operation, not permanent but after the operation has been performed, particularly so in fixed squint, orthoptic treatment may be very useful. After the operation for fixed squint, in the

first class there may exist that condition described by Von Graefe as an "antipathy to binocular vision," in other words, while the obvious conditions may show complete parallelism of the visual lines, yet true binocular vision is not present and the patient will not see with one eye. We must remember that the two eyes should act physiologically as one eye, or cyclopean and unless there is a decided muscular anomaly they must act together and see together. Hence we may say it is a natural function of the eyes that causes a non-operated eye to show the effects of an operation on the other eye in the fields of version and fusion, without regard to the innervation that may stimulate any one of the ocular muscles to action. This has been denied by other writers but it seems to me that I have found it very noticeable in many of my cases after an operation.

Now to correct this "antipathy" or to improve the fields of motion by which we may stimulate the physiological part of vision to become established, is frequently a very perplexing problem and one that will frequently meet with failure. We may operate and the tests may show perfect movement of the eyes in the field of version, but fusion of images, that automatic part of the act of vision, cannot be produced. To restore parallelism and produce perfect binocular vision is the ideal result to be obtained. In some cases we may succeed; in others, the most persistent efforts will only result in failure; yet the object to be attained is, none the less, necessary and useful to the patient, hence all reasonable efforts should be instituted for its attainment. I have had cases where the results seem to be excellent; there was no evidence of squint, yet I could not develop the slightest degree of binocular vision. There was no field of fusion whatever. Such cases I have generally found in squint of the first class, that is, squint associated with

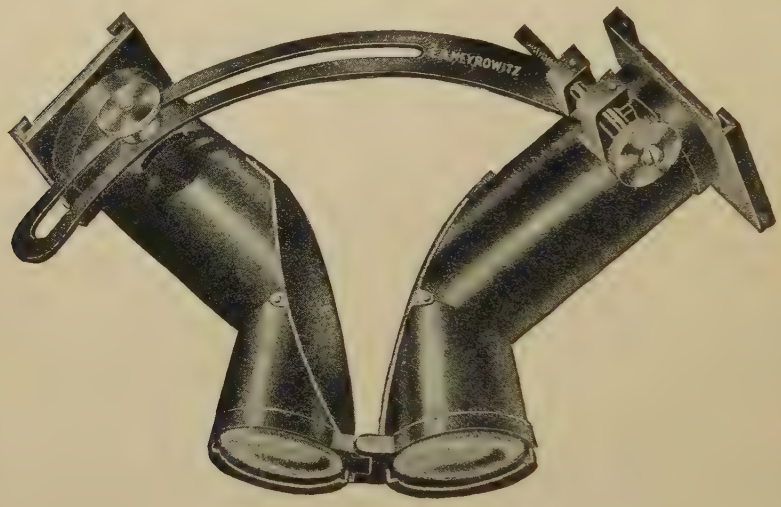
congenital amblyopia. If this condition exists, I am satisfied with a good cosmetic result, and make no effort to bring about binocular vision; conversely, if we have squint of the second class, in which we find fairly good vision in each eye 20–70, or better, then I make every reasonable effort, that the patient will permit, to restore or produce binocular fixation and fusion. The vision in amblyopia ex anopsia may be frequently improved by treatment, and with the improvement of the vision we may find a decided improvement of the fusion force with eventually binocular vision. Habit must play a very important rôle in these cases. This may be overcome and corrected by persistent effort. If then, our operation has been performed, the vision is fairly good and improving — what procedures are indicated?

Drugs will be of little or no value now, except so far as we may improve the general condition of the bodily health. It goes without saying that in all cases the refraction must be corrected as the first consideration. Exercise of the individual muscles is frequently of service. This is done by the use of prisms, with the apex placed over the insertion of the muscle to be developed. Constant exercise of this kind will in many cases improve the fusion force, and may be continued as long as the results show any improvement.

Hansel and Reber (page 108) claim that by prism exercise the power of adduction may be carried to 100° inside of eight or ten weeks. I have never been able to bring about such a happy result in my cases, and it seems to me that a prism of 100° held properly before the eye is very close to the "limit angle," unless two prisms are used of 50° each, placed base outward, and if so, I do not think we are stimulating the fusion force of the eyes, but simply the convergent center, the same as if the object were brought nearer to the eyes, up to the near point.

Closing one eye having the better vision, by bandage or pad for five or ten minutes every day, may improve the vision of the amblyopia from disuse, though I do not like this procedure after an operation.

The effort to blend the pictures of a stereoscope is useful, and tend to improve the function of the third dimension if there is any tendency to the use of both eyes, as in this way we stimulate fusion and improve the mental processes of the act of vision. Suitable pictures for this purpose can be procured from Mr. E. B. Meyrowitz of this city. Furthermore, in the after-treatment of all these cases of latent and fixed squint I think the amblyoscope of Worth, England, is a very useful instrument.



WORTH'S AMBLYOSCOPE.

I cannot fully approve of all Worth claims for this instrument to bring about fusion, particularly before an operation, as his statistics of a large number of cases do not show any larger percentage of corrections than that by the use of glasses; but in squint of the second class,

where we have a lateral deviation or a latent squint, then after an operation I think this instrument a very valuable one. A full description of its use may be found in Worth's monograph on squint, lately issued. This is an excellent method of stimulating the habit of blending the images on each retina — one of the most interesting psychological functions of nature — when they tend to separate in squint, and for the purposes of orthoptic training for the establishment of binocular vision it is very useful. This instrument consists of two tubes, each forming an angle of 120° with two oval mirrors placed at the apex of the angle. At the distal ends of the tubes are grooves for holding the glass slides with the pictures drawn on them. At the proximal ends are two convex lens whose focal distance is situated at the pictures, so that the rays entering the eyes will pass as from infinity. At these ends we also find grooves or slots to hold any glasses necessary to correct an error of refraction. These tubes are joined by a hinge at the proximal ends, so that they may be adjusted to any angle of squint. The object slides, as the bird and the cage, are simply to teach the child to see with each eye the image formed on each retina and then by moving the tubes the bird can be made to enter the cage. The other slides, having only parts of the pictures on them, with certain similar parts very prominent, must be blended to form a perfect picture. The slides with the circles are to be blended and should also give the effect of solidity or the "third dimension." When these images can be readily blended the tubes are to be moved closer together to stimulate convergence and apart for divergence as long as the images do not separate. By this process, Worth claims, we gradually increase the power of the eyes to blend the images on the retina and stimulate the retina to its natural function from the increase of the field of fusion. This method of treatment

has some advantages over that of the stereoscopic pictures, as we can produce a blending of the pictures at any angle of squint before an operation, or that may remain after the operation has been performed. Then by the gradual changing of the position of the pictures we stimulate the function of blending until binocular vision may be established. That this practice requires an immense amount of patience on the part of the surgeon and the patient goes without saying, at the same time, the ultimate result of binocular vision that may be attained is well worth a careful and serious effort. If one eye is amblyopic, then when using this instrument we may reduce the illumination of the slide before the eye with the better vision, so as to stimulate the vision of the amblyopic eye, and in this way increase its power to blend the image with that of the other eye. The illumination of an object has a great influence on the power to fuse similar images as well as the power to see, but as to the value of this instrument in the correction of squint, of the second class, with fairly good vision in each eye, I have had no experience and Worth's statistics seem to me about the same as others claim from the use of glasses that will correct the refractive error. In the after treatment of squint I think the amblyoscope will be very useful, as this exercise must have some influence on the blending of images on the retina of each eye, as the tubes enable the patient to have single vision when the visual lines are deviated, but I do not like the term fusion, as this apparatus does not show nor measure the field of fusion in any way.

Finally, all cases of muscular asthenopia, and even fixed squint, may be improved by an active life in the open air, exercise and diet, but we must consider all these cases from their individual lives. We cannot advise all our cases of muscular asthenopia to ride horseback or take a trip to Europe, but we must consider their environment

and the needs of their daily life. We cannot take many of them from their daily occupations and duties, and so our mode and method of treatment must be adjusted to the circumstances of our patients.

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